

Epidemics in Networks

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Introduction

Model building

Mass Action SIR model

SIR epidemics in Static Networks

Static Networks II

SIS Diseases

SIR Epidemics in Dynamic Networks

Limitations

Introduction

Why Model Infectious Diseases?

A motivating example



Why Model Infectious Diseases?

Much of the mathematics used in Public Health is statistics. Why do we need dynamic models for infectious diseases?

- If we know someone's exposure to some toxin, then we can use statistics to determine the probability of developing some illness.
- It's straightforward then to determine the impact of some policy that reduces exposure to the toxin.



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- It's straightforward then to determine the impact of some policy that reduces exposure to the toxin.
- Reducing exposure by 1/2 reduces bad outcomes by 1/2.
- Statistical models can answer our questions.

Mechanistic modeling

This approach doesn't work with infectious disease.

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- So your vaccination reduces my risk, which in turn reduces the risk of my partners.
- We need a mathematical model to allow us to determine how these nonlinear feedback mechanisms play out.
- Similar issues may arise for behaviors such as smoking.

Important questions

If a new disease may emerge, policy makers are likely to be very interested in the following questions:

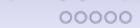
- Is it possible for an epidemic to occur?
- How likely is it that a single introduction leads to a large epidemic?
- How many people would be infected?
- How can we predict the time-course of the epidemic?

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We build mathematical models to give us answers to these questions.



How complex should a model be?

Modeling \neq mountain climbing

$$\dot{S} = -\beta kIS$$

$$\dot{I} = \beta kIS - \gamma I$$

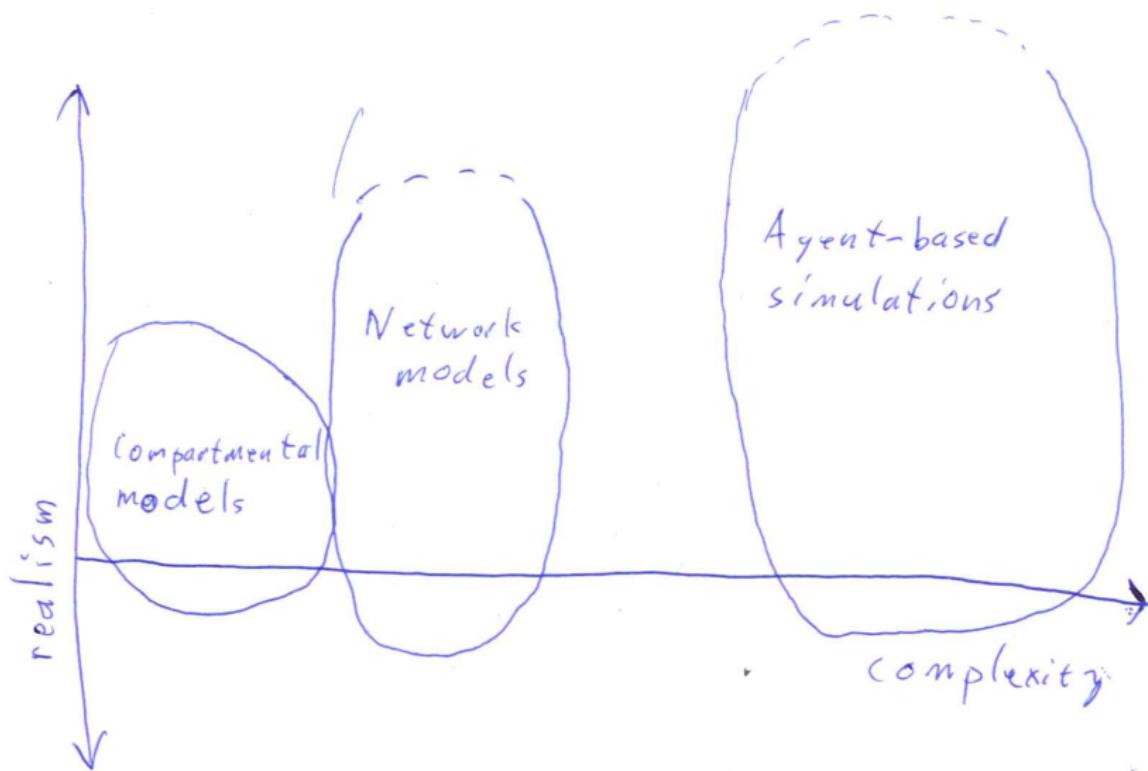
$$\dot{R} = \gamma I$$

 \neq 

- We should not incorporate some detail into our model simply because it's there.
- We should only include things that could affect the decisions policy makers make.
- Increased complexity is only worthwhile if it will improve our policy.



Modeling options





What do we need to model?

- The properties of the disease relevant for transmission.
- How contacts that can transmit disease are structured in the population.

Introduction

Why Model Infectious Diseases?

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A thought experiment

Consider a population into which HIV has just been introduced.
Assume that

- On average individuals in the population have 4 sex acts a week.
- The transmission probability per sex act is 0.01.

Our goal is to reduce the early spread, so we ignore birth/death (including disease-caused death). Note, we measure time in years.

Homogeneous well-mixed assumptions aka “mass action” (a null model)

If all people have the same contact rate and choose each partner randomly, then early in the spread:

- An average infected individual has about 200 sex acts per year and so causes ≈ 2 infections per year.
- The rate of increase of I , the infected proportion, is $\frac{d}{dt}I = 2I$.

Heterogeneous mixing

If different people have different contact rates, but still choose each partner randomly:

- The people with the highest contact rate will tend to be infected earlier and cause more infections.
- Early growth is enhanced.
- Note: the people with lower contact rates may escape infection much longer than in the mass action model. Higher early growth may not translate into a larger epidemic.

Mongomous Partnerships

Assume partners are not chosen randomly each time — each individual has one partner who does not change.

- The epidemic cannot spread.

Serial Monogamy

Assume monogamous partnerships, but partnerships change in time.

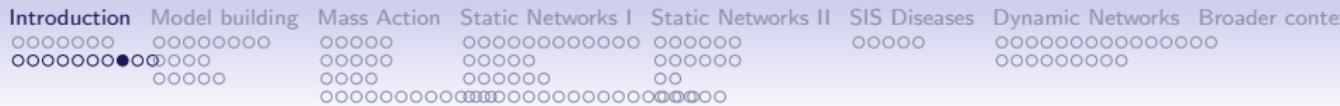
- Spread is no longer limited just by low transmission probability, but also duration of partnerships.
- If partnership duration small compared to time required to transmit, we can treat this as mass action, ignoring partnerships.
- If partnership duration large compared to time required to transmit, we can treat this as mass action, with the effective rate of transmission coming from partnership dissolution.¹

¹I hypothesize. Showing this and exploring further would probably be a JMB/JTB publication.

Overlapping partnerships

Assume some/all individuals have several partnerships which overlap in time.

- It's probably faster growth than serial monogamy, but slower than if every contact is with a new individual.
- There is a large controversy over "concurrent relationships" and their role in HIV transmission. I believe it is in large part fueled by an absence of clear models.
- See the first 6 articles in AIDS and Behavior from Feb 2010 to get an idea of how contentious this is.



The list goes on

...



What can modeling tell us?

- Very crude models can tell us which growth rates will be larger
- More complex models can tell us
 - the early growth rates
 - the long-term dynamics
 - the relative effectiveness of different interventions
 - or, if the details added are insignificant, nothing that's not in the simple models.

How good does our model need to be?

The complexity of the model only needs to be good enough to answer the question. Often ignoring some of the complexity in the population won't change the policy recommendation.

- Is it good to promote condom use?
- Is it good to target people with higher contact rates when promoting condoms?
- Is it good to target people with higher contact rates if the resources needed to do that mean fewer total people are reached?
- Is it worth taking resources away from promoting condom use to convince people to not have overlapping partnerships?

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More nuanced questions require more complete models.

Model building

Model options

Basic mathematical principles

Empirical networks



What kinds of models can we use?

- Careful thought (really just a simple mathematical model)
- Mathematical model
- Simulation



Simulation vs Mathematical models

Usually simulation is more flexible, but a mathematical model is easier to work with.

My (biased) opinion:

If you can't create a simulation that matches your mathematical model, then either:

- You are a very good mathematician working on a very difficult problem, and you've put in a lot of effort to determine the limits of your model or
- You are doing something wrong.

You should know what assumptions are required to derive the equations you are using, and you should pay particular attention to which assumptions are violated by reality.

Some things to consider including in a model

- Heterogeneous contact rates
- Partnership duration
- Clustering (my friends are friends of each other)



Heterogeneity in contact rates

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- Tends to decrease epidemic sizes:



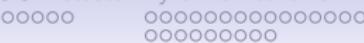
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- Can be as simple as a core group and a general population, or can have a wide distribution of behaviors.
- This is discussed in many papers, including [1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12]

Partnership duration

- Individuals may transmit more than once to the same partner or may transmit back to their infector.
- If the expected time to transmission is significantly larger than the expected duration of a partnership, there are very few “wasted” transmissions and we can ignore duration.
- If not, then it may be necessary to include duration in the model.
- Longer partnership durations tend to slow epidemic spread and reduce final sizes.
- Most existing models either assume duration is zero or infinite.
- [13, 14, 15, 16, 17, 18, 19, 20, 11, 12]



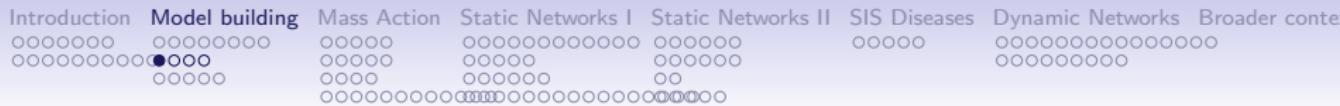
Clustering

- Clustering tends to reduce the rate of spread because many transmission chains intersect.
- We can estimate whether triangles are important by calculating the density of triangles and the probability that all edges of a triangle would transmit. If many transmissions are “wasted” then clustering has a significant effect.
- Finding good mathematical or simulation models to incorporate clustering is a difficult and largely open problem.
- [21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37]



Basic approach to creating a mathematical model

- Consider the population and disease being modeled.
- Determine how you would simulate the epidemic:
- Find equations that capture the rules of the epidemic (as close as possible).
- Note: there may be more than one reasonable way to model the epidemic.

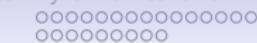
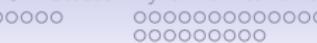


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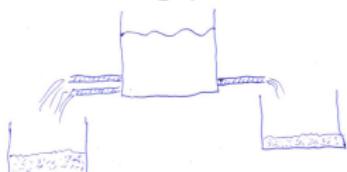
Empirical networks



Swimming pool analogy

I'll refer to the "swimming pool analogy" several times.

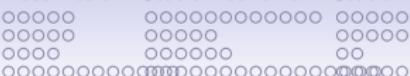
- Imagine that there is a source of water, from which two swimming pools are filled.



- If both pools start empty, and at all times one receives c times as much flow as the other, then it will always have c times as much water in it.
- If the source is additionally finite, then when all the water has left the source, the first will have $c/(1 + c)$ of the initial water and the second will have $1/(1 + c)$ of the initial water.

Basic differential equations

- We will frequently be interested with the rate the quantity of something changes in time.
- Typically this will involve the rate that thing is created minus the rate it is destroyed.
- We use a dot to denote the change in something in time. For example, \dot{S} is the rate that S changes in time.
- Mathematically, \dot{S} is the time derivative of S .



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Basic probability with independent events

- We will frequently be interested in the probability that at none of n events haven't happened.
- Assume each event is independent of the other, and p_1, p_2, \dots, p_n are the probabilities of each event.
- Then the probability none of the events has happened is

$$(1 - p_1)(1 - p_2) \cdots (1 - p_n)$$

- The probability at least 1 has happened is

$$1 - (1 - p_1)(1 - p_2) \cdots (1 - p_n)$$

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- Romantic networks [45]

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- However, we can change our focus to locations (or other collections of individuals) with edges representing individuals that go between different locations.
- Typically we assume some simple mixing within locations and look at how disease spreads from location to location.

Sample location-location networks

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- Seasonal population movements [51]: study of seasonal population movements for malaria control (phone data, census, satellite imagery).

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- South Africa: Simulation by George Seage's group at HSPH for HIV transmission (≈ 6 million?)

Mass Action SIR model

Basic SIR models

Epidemic Probability

Epidemic Final Size

Epidemic Dynamics

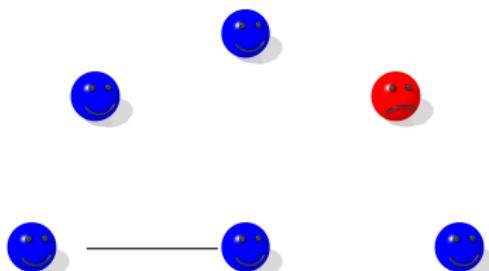
SIR Models

We begin with “SIR” diseases.

- Most individuals begin susceptible.
- Contact with infected individuals may cause susceptible individuals to become infected
- Infected individuals recover and are immune.

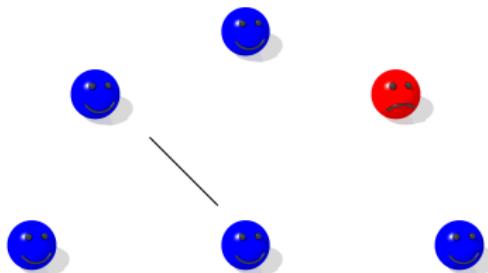


The basic SIR model



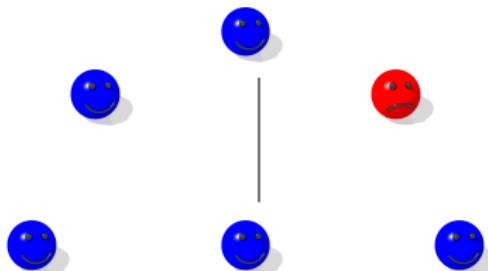
- Individuals begin **susceptible**,
- become **infected** from contacting infected individuals,
- and eventually **recover** with immunity.

The basic SIR model



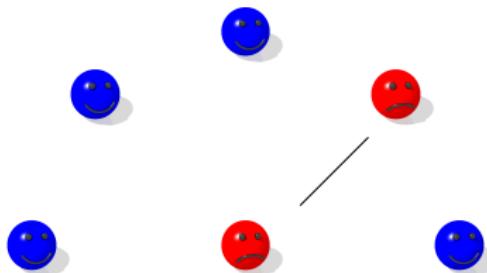
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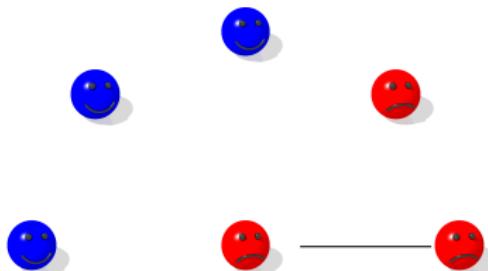
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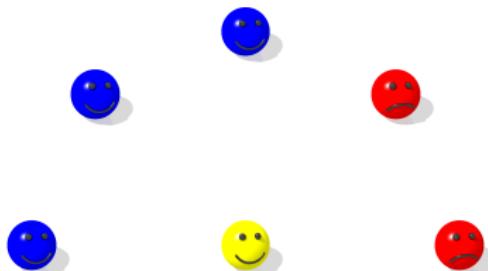
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Population divided into Susceptible, Infected, and Recovered individuals, $S + I + R = 1$.

- “Mass Action” mixing
 - Everyone has exactly k contacts.
 - At each moment in time, those k contacts are with new people.
- Infection is transmitted at rate β per contact.
- Recovery occurs at rate γ .

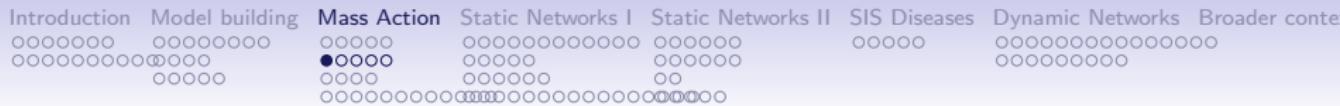
(alternate interpretations exist)

Possible outcomes

If we introduce an infection to a population:

- If conditions are unfavorable for the disease, there may be a small outbreak but the disease quickly dies out.
- Even if conditions are favorable for the disease, we might simply be lucky and the chains of infection die out without becoming large.
- If conditions are favorable, there is a nonzero chance that the outbreak becomes large — we call this an epidemic.

We will look at the probability of an epidemic, the size of an epidemic, and the time-course of an epidemic.



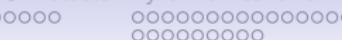
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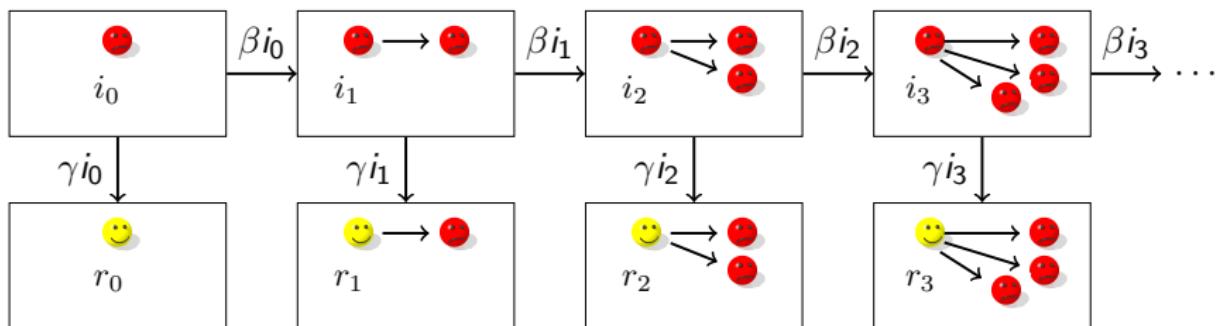


Epidemic probability for mass action

Assume a large population. Let \mathcal{P} be the epidemic probability.

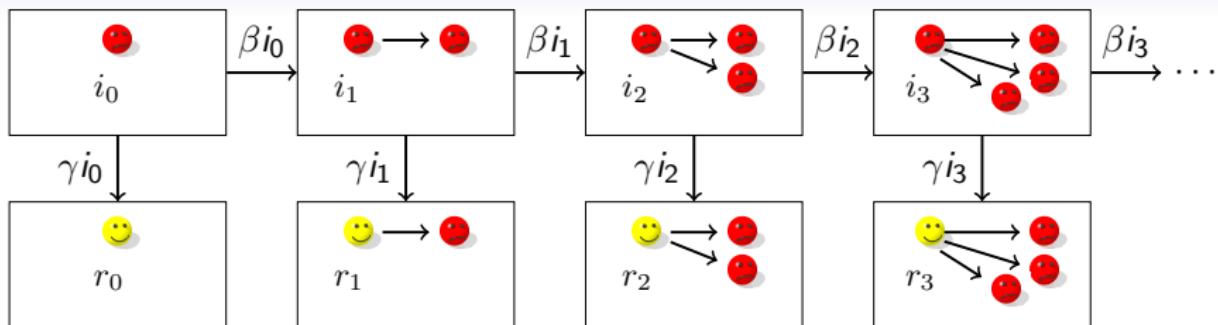
- The probability an epidemic occurs is the probability the first infection has many “descendents”.
- This is the probability that the first infection directly infects at least one individual who has many descendents.
- It is easier to calculate the probability of not starting an epidemic: this is the probability that no “daughter” will start an epidemic.
- Because each individual has the same behavior and constantly selects new partners, the probability a given daughter would start an epidemic (if infected) equals the probability the index case does.

Calculating \mathcal{P} .

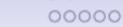


Consider an individual u who becomes infected at time $t = 0$.

- Let $i_m(t)$ be the probability u is infected at time t and has infected m individuals.
- Let $r_m(t)$ be the probability u has recovered by time t and infected m individuals.



- By swimming pool analogy, the probability the first thing an infected individual does is transmit is $\beta/(\beta + \gamma)$.
- The probability the first thing is a recovery is $\gamma/(\beta + \gamma)$.
- If the first n things are transmissions, then the probabilities for the $n + 1$ st thing to be a transmission or recovery are $\beta/(\beta + \gamma)$ and $\gamma/(\beta + \gamma)$.
- So the probability the first m events are transmissions and the $m + 1$ st event is a recovery is $r_m(\infty) = \left(\frac{\beta}{\beta+\gamma}\right)^m \frac{\gamma}{\beta+\gamma}$



From previous slide $r_m(\infty) = \left(\frac{\beta}{\beta+\gamma}\right)^m \frac{\gamma}{\beta+\gamma}$

The probability of no epidemic is

$$\begin{aligned}
 1 - \mathcal{P} &= \sum_{m=0}^{\infty} r_m(\infty)(1 - \mathcal{P})^m \\
 &= \frac{\gamma}{\beta + \gamma} \sum_{m=0}^{\infty} \left(\frac{\beta}{\beta + \gamma} (1 - \mathcal{P}) \right)^m \\
 &= \frac{\gamma}{\beta + \gamma} \left(\frac{1}{1 - \frac{\beta(1-\mathcal{P})}{\beta+\gamma}} \right) \\
 &= \frac{\gamma}{\gamma + \beta\mathcal{P}}
 \end{aligned}$$

You didn't believe you'd need to sum infinite series after Calculus, did you?



Mass Action SIR model

Basic SIR models

Epidemic Probability

Epidemic Final Size

Epidemic Dynamics

First an observation from Survival Analysis:

- Consider an initially susceptible test individual u .
- Let $\xi(\infty)$ be the cumulative hazard of infection for u over the course of the epidemic. It is the expected number of times that u would receive infection.
- The probability that u is still susceptible at the end of the epidemic is $e^{-\xi(\infty)}$.

We're going to focus our attention on $\xi(\infty)$.



Assume $S(0) \approx 1$ and $R(0) \approx 0$, and there are $N \gg 1$ individuals

- The probability that u is eventually infected is $R(\infty)$.

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$$1 - R(\infty) = (1 - 1/(N - 1))^{(N-1)\mathcal{R}_0 R(\infty)} \approx e^{-\mathcal{R}_0 R(\infty)}.$$



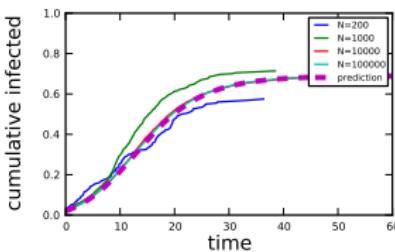
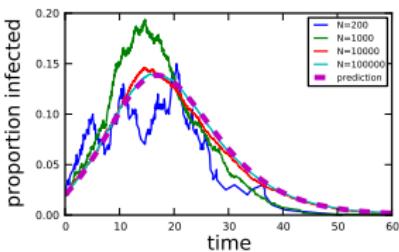
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.
- Our final size is thus

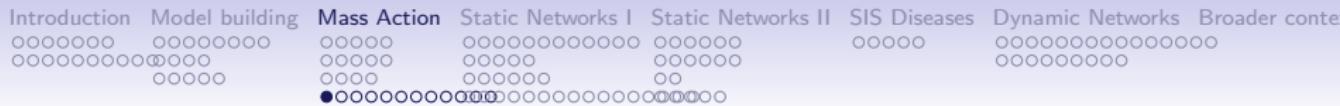
$$R(\infty) = 1 - e^{-\mathcal{R}_0 R(\infty)}$$

Tangential comments

- The derivation we just did requires no dynamic equations.
- It should be possible to derive final size relations without dynamic equations because simulations show that the final size converges for smaller population sizes than the dynamics do.



- It also doesn't rely on constant transmission or recovery rates — only that a random infected individual transmits to u with probability \mathcal{R}_0/N .
- This argument directly gives most of the results in [56]. It allows more general infection processes than we've considered [57].



Mass Action SIR model

Basic SIR models

Epidemic Probability

Epidemic Final Size

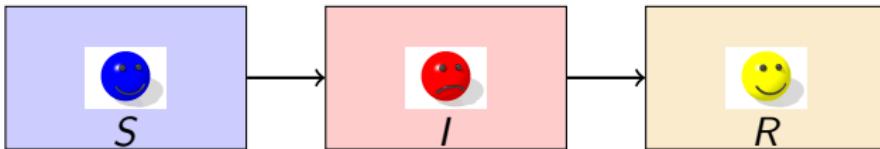
Epidemic Dynamics

- In calculating the epidemic dynamics, we'll follow the usual derivation that many may have seen already.
- Then we'll revisit it and derive a different set of equations using a method very much like what we just did for the final sizes.
- The equations are equivalent.



The mass-action model

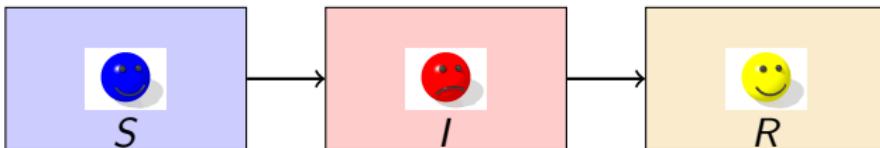
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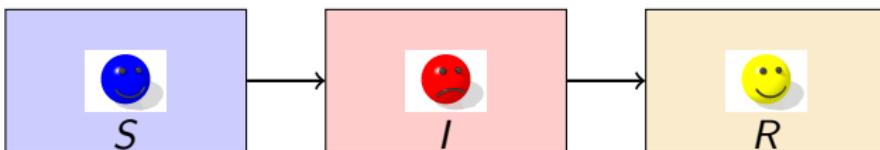


- Because recovery is at rate γ per individual, the flow from the I compartment to the R compartment is γI .



The mass-action model

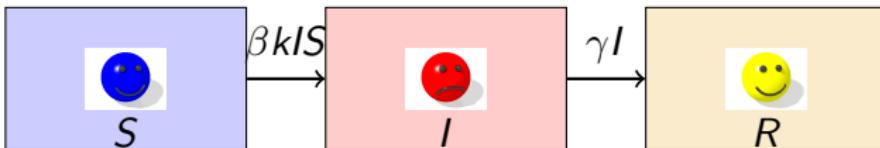
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- Each infected individual transmits at rate βk . The recipient of a transmission is susceptible with probability S . The S to I flow is $\beta k I S$.

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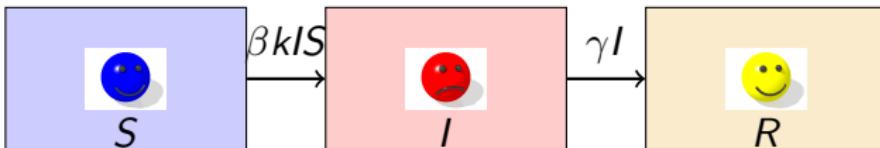


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- Because recovery is at rate γ per individual, the flow from the I compartment to the R compartment is γI .
- Each infected individual transmits at rate βk . The recipient of a transmission is susceptible with probability S . The S to I flow is $\beta k S I$.
- Resulting equations:

$$\dot{S} = -\beta k S I, \quad \dot{I} = \beta k S I - \gamma I, \quad \dot{R} = \gamma I$$

An alternate perspective on the Mass Action model

- We're going to approach the same model from a perspective, much like we used to derive the final size relation.
- We'll arrive at different equations, but they are equivalent.
- This different perspective will be useful for understanding epidemic spread in networks.



First we revisit our observation from Survival Analysis:

- Consider an initially susceptible test individual u .
- Let $\xi(t)$ be the cumulative hazard of infection for u since $t = 0$. It is the expected number of times that u would receive infection.
- The probability that u is still susceptible at time t is $e^{-\xi(t)}$.

We're going to focus our attention on $\xi(t)$.



finding $\xi(t)$

- All individuals are the same, so $\xi(t)$ is the expected number of transmissions any individual has received since $t = 0$.
- $N\xi(t)$ is the total number of transmissions.
- Since each transmission has a source and a recipient, $\xi(t)$ is also the expected number of transmissions a random individual has caused.



finding $\xi(t)$

- So $\xi(t)$ is the average number of transmissions caused since $t = 0$.
- Since infected individuals transmit at rate βk , we find that $\dot{\xi} = \beta k I$.
- Infected individuals recover at rate γ , so $\dot{R} = \gamma I$.
- The rate R increases is $\gamma/(\beta k)$ times the rate ξ increases.
- By the swimming pool analogy (infinite source) the amount R has increased is $\gamma/(\beta k)$ times the amount ξ has increased.



finding $\xi(t)$

We arrive at

$$\dot{\xi} = \beta k I$$

$$S(t) = S(0)e^{-\xi(t)}$$

$$R(t) = R(0) + \frac{\xi(t)}{\mathcal{R}_0}$$

$$I(t) = 1 - S(t) - R(t)$$

where $\mathcal{R}_0 = \beta k / \gamma$.

If we want, we can reduce this to

$$\dot{\xi} = \beta k [1 - S(0)e^{-\xi(t)} - R(0)] - \gamma \xi(t)$$

This derivation is at the end of [57]

How does:

$$\dot{\xi} = \beta k l$$

$$S(t) = S(0)e^{-\xi(t)}$$

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compare with the usual SIR equations?

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compare with the usual SIR equations?

$$\dot{R} = \dot{\xi}/R_0 = \beta k l / R_0 = \gamma I$$

$$\dot{S} = S(0)(-\dot{\xi}e^{-\xi}) = -S(0)\beta k l e^{-\xi} = -\beta k l S$$

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How does:

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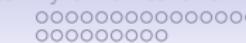
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$$\dot{I} = -\dot{S} - \dot{R} = \beta k I S - \gamma I$$

So we get the same equations.



An immediate advantage - an alternative final size derivation

- We have

$$\dot{\xi} = \beta k [1 - S(0)e^{-\xi(t)} - R(0)] - \gamma \xi(t)$$

- At the end of the epidemic $\dot{\xi} = 0$.
- So $\xi(\infty) = \frac{\beta k}{\gamma} [1 - S(0)e^{-\xi(\infty)} - R(0)]$
- Assuming $S(0) \approx 1$ and $R(0) \approx 0$

$$\xi(\infty) = \mathcal{R}_0 (1 - e^{-\xi(\infty)})$$

- If $R(0) = 0$, then $R(t) = \xi(t)/\mathcal{R}_0$. So our equation becomes

$$R(\infty) = 1 - e^{-\mathcal{R}_0 R(\infty)}$$



Lessons for our networks

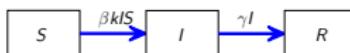
- We've derived a single differential equation that governs the standard mass action SIR model.
- The way to do this derivation is to focus on the probability a random individual is still susceptible.
- We will continue this approach as we consider SIR diseases spreading in a network.



Why use the Mass Action model?

When there is variation in contact rates or contacts have long duration, mass action model assumptions are false. Why use them?

- Simple equations
- + Simple graphical description
- = Simple interpretation

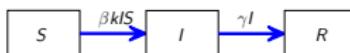




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Does cost/effort of more accurate model give improved policy recommendation?



SIR epidemics in Static Networks

More Contact Structure

Epidemic Probability

Final Size

Dynamics

Network Assumptions

We begin with basic assumptions about contact structure.

- The number of partners an individual has is its “degree” k .
 - k varies from individual to individual.
 - $P(k)$ is the proportion of the population with degree k .
 - We seek the simplest model that satisfies these assumptions.
 - Partners are randomly chosen.
 - The network connections do not change (static network).
This assumption is appropriate if the timescale of the epidemic is less than the typical partnership duration.

Constructing a Configuration Model Network

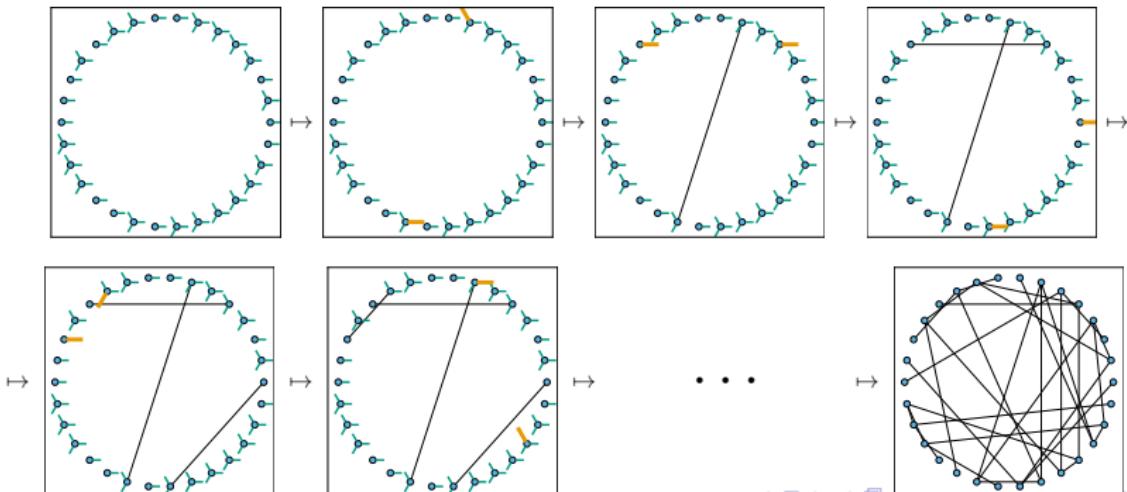
Given a $P(k)$, the concept for the network construction is as follows:

- Assign each node a degree k , and give it k “stubs”.
- Choose pairs of stubs at random, and join them.
- Continue until done.

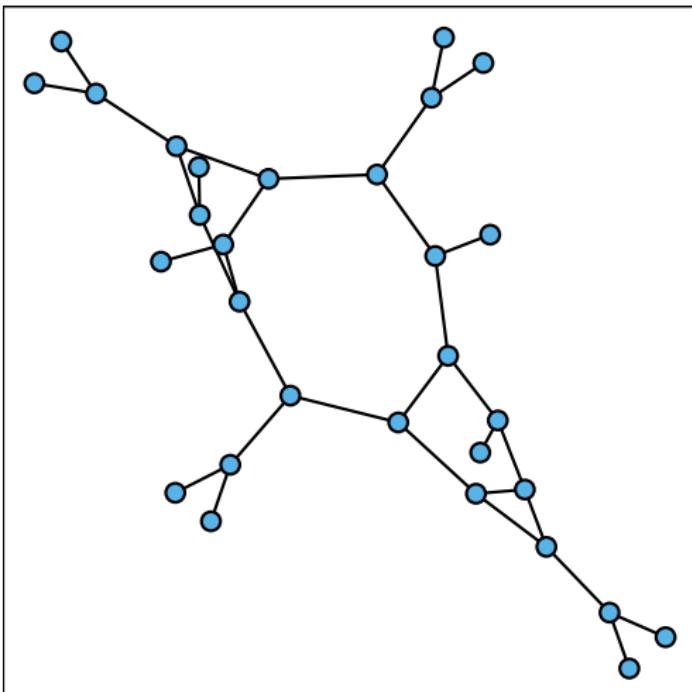
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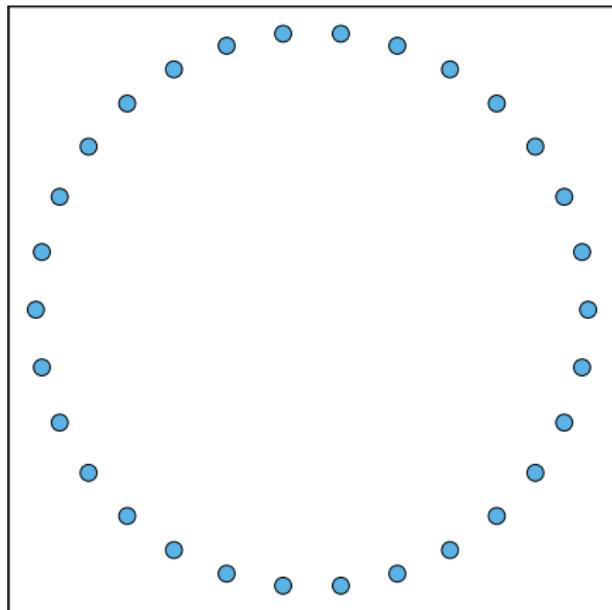


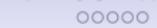
Final result



Size Bias

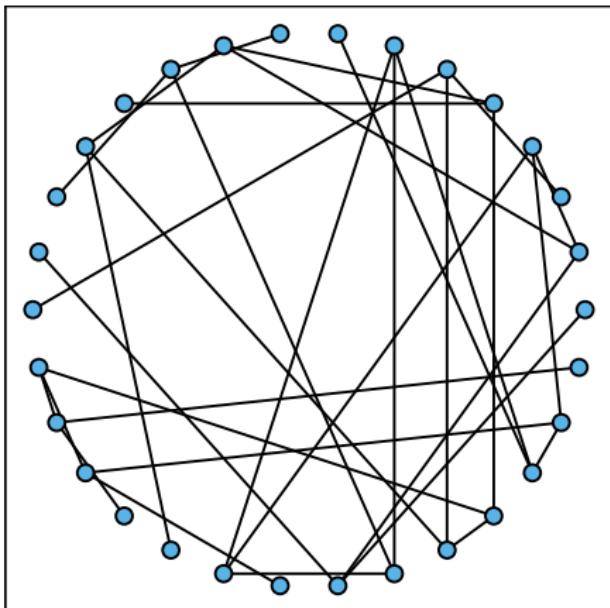
Do your friends have more friends than you do (on average)?





Size Bias

Do your friends have more friends than you do (on average)?



Size Bias

- A random individual has degree k with probability $P(k)$
- What about a random partner? What is the probability $P_n(k)$ a partner has degree k ?
- Because of how partners are selected, a random partner is likely to have higher degree than a random individual [58, 59].
- In fact $P_n(k) = kP(k)/\langle K \rangle$ where $\langle K \rangle$ is the average degree.
- Note that the degrees of a random individual and a random neighbor of a random individual have different distributions, but a random neighbor and a random neighbor's random neighbor are both from $P_n(k)$.

Size Bias

I cannot stress enough that if $P(k)$ is the probability a random individual has k partners, then

$$P_n(k) = kP(k)/\langle K \rangle$$

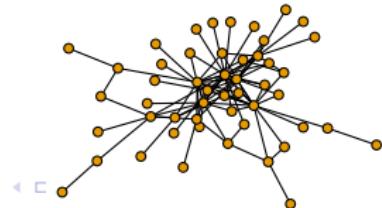
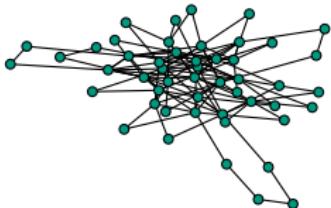
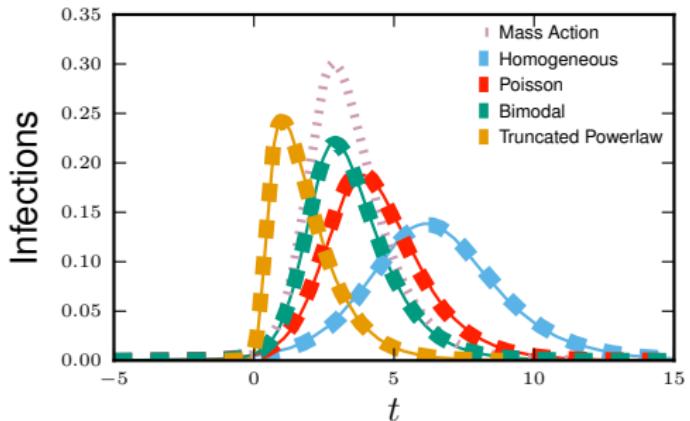
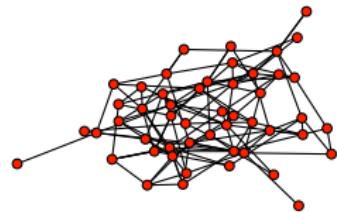
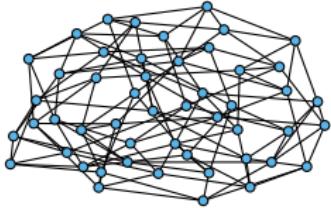
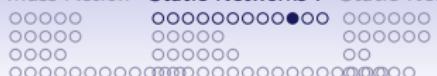
is the probability a random partner has k partners.

Disease biology

- We assume Infection spreads along each edge at rate β .
- An infected individual recovers at rate γ .
- By the pool analogy again: The probability an infected individual transmits to its partner before recovering is $\beta/(\beta + \gamma)$. We'll refer to this as T later.



Is the average degree enough to predict epidemic behavior?



Why the discrepancy with Mass Action and one another?

There are two effects going on:

- Individuals with high degree tend to become infected first. Then they infect more additional contacts. This increases early growth.
- Contacts have long duration: when an individual infects a contact, s/he loses a susceptible neighbor.
- Epidemic probability, final size, and time evolution are all affected by partnership duration and degree heterogeneity.



Can we fix the mass action model cheaply?



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Calculating epidemic probability

- To calculate the probability of an epidemic we imitate the calculation for the Mass Action Model.

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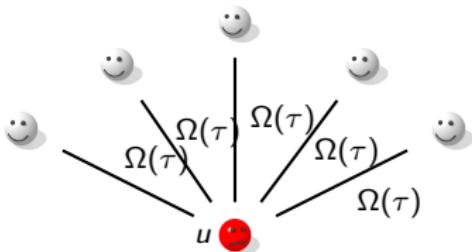


Calculating epidemic probability

- To calculate the probability of an epidemic we imitate the calculation for the Mass Action Model.
- We assume that the initial infection is chosen randomly from the population. The probability it does not cause an epidemic is equal to the probability none of its “daughters” causes an epidemic.
- This is more difficult because each time an individual transmits, it loses a neighbor, so its transmission rate is reduced.



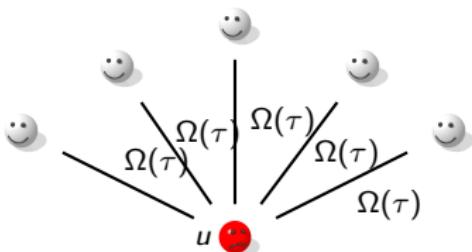
Calculating epidemic probability



$$\Omega(\tau) = P(u \text{ does not transmit to a neighbor} | \tau) + P(u \text{ transmits, but neighbor doesn't lead to an epidemic})$$



Calculating epidemic probability



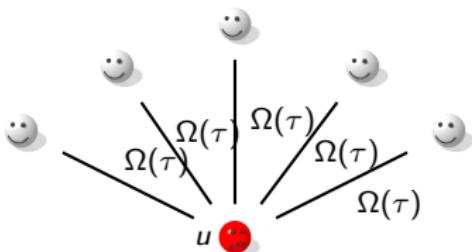
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Probability a random degree k index case whose infection duration is τ does not start an epidemic is

$$\Omega(\tau)^k$$



Calculating epidemic probability

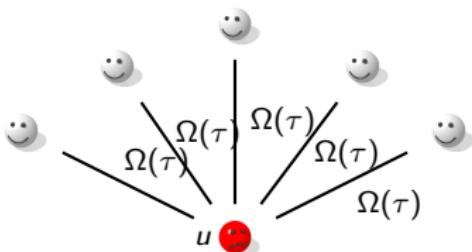


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Calculating epidemic probability



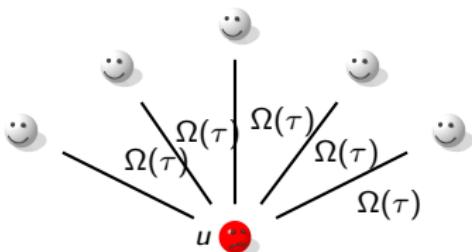
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$$1 - \mathcal{P} = \int_0^\infty \gamma e^{-\gamma \tau} \sum_k P(k) \Omega(\tau)^k d\tau$$



Calculating epidemic probability



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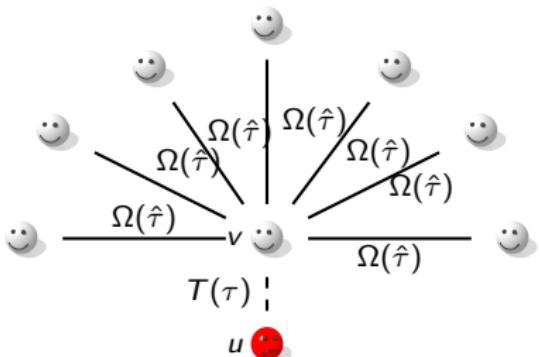
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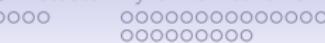
$$1 - \mathcal{P} = \int_0^\infty \gamma e^{-\gamma\tau} \sum_k P(k) \Omega(\tau)^k d\tau = \int_0^\infty \gamma e^{-\gamma\tau} \psi(\Omega(\tau)) d\tau$$

where

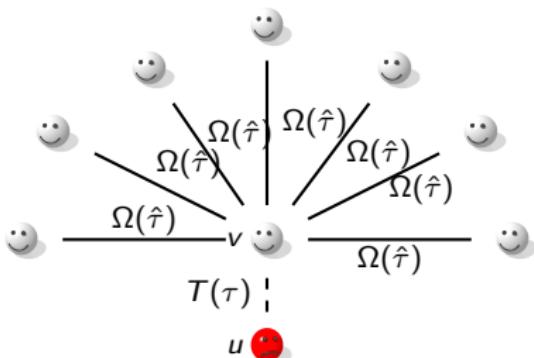
$$\psi(x) = \sum_k P(k) x^k$$

Finding Ω



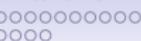


Finding Ω

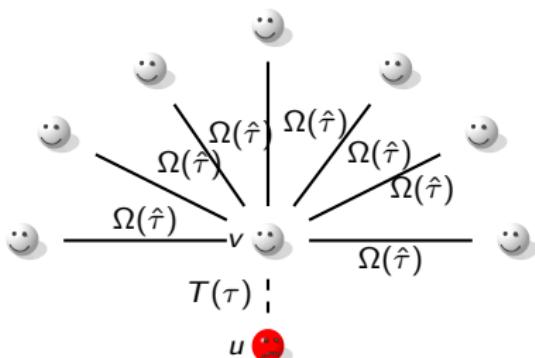


Probability a random partner of the index case having degree \hat{k} whose infection duration is $\hat{\tau}$ does not start an epidemic is

$$[1 - T(\tau)] + T(\tau)\Omega(\hat{\tau})^{\hat{k}-1}$$



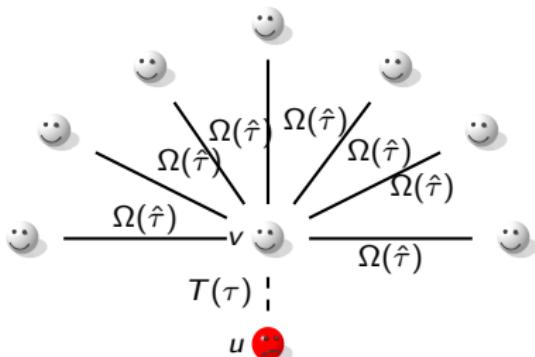
Finding Ω



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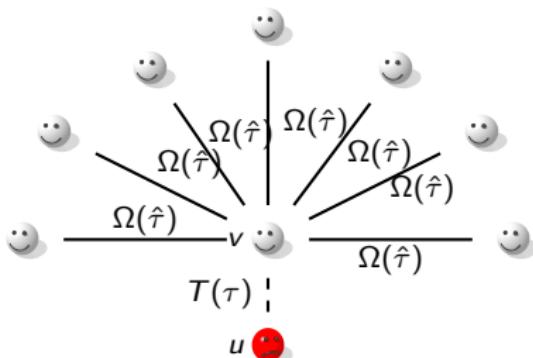
Finding Ω



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$$\Omega(\tau) = [1 - T(\tau)] + \int_0^{\infty} \gamma e^{-\gamma \hat{\tau}} \sum_{\hat{k}} P_n(\hat{k}) T(\tau) \Omega(\hat{\tau})^{\hat{k}-1} d\hat{\tau}$$

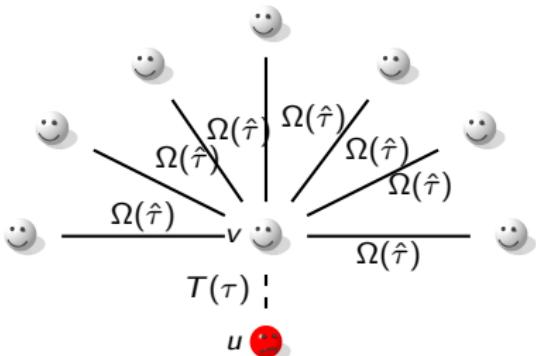
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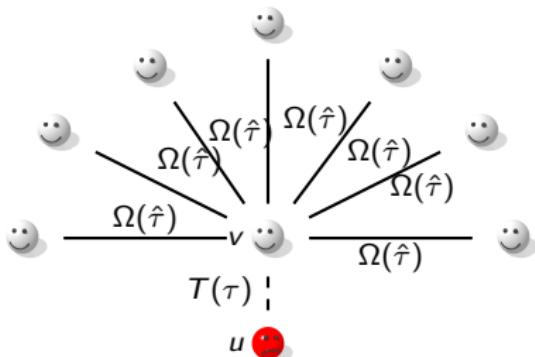
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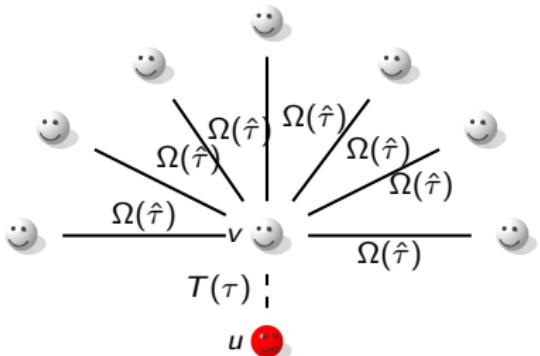
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Finding Ω



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Calculating epidemic probability

We arrive at

$$1 - \mathcal{P} = \int_0^\infty \gamma e^{-\gamma\tau} \psi(\Omega(\tau)) d\tau$$

$$\Omega(\tau) = 1 - T(\tau) + T(\tau) \int_0^\infty \gamma e^{-\gamma\hat{\tau}} \frac{\psi'(\Omega(\hat{\tau}))}{\psi'(1)} d\hat{\tau}$$

In general we can only solve this numerically, but it is straightforward.



SIR epidemics in Static Networks

More Contact Structure

Epidemic Probability

Final Size

Dynamics



Our derivation here is a hybrid of [2, 60, 61]

The final size of an epidemic in a network

- Consider a randomly chosen test individual u in the population.

²Details about this are in [11, 62].

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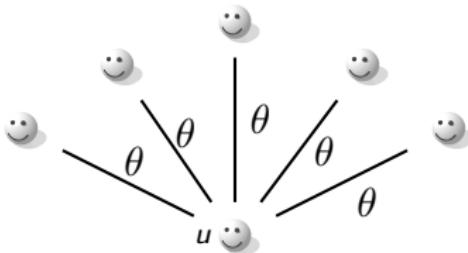
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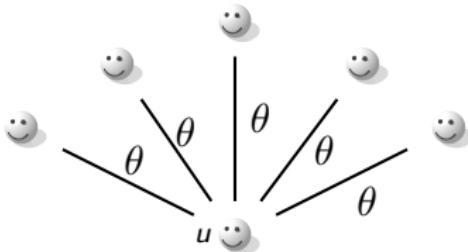


Finding S



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Finding S

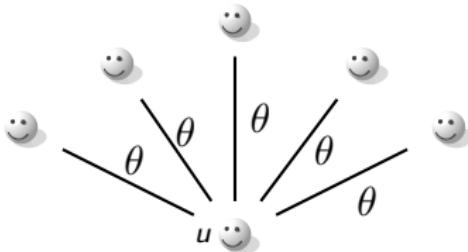


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Probability a random degree k test individual remains susceptible is

$$\theta^k$$

Finding S

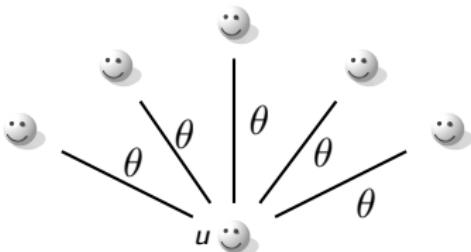


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Probability a random **degree- k** test individual remains susceptible is

$$S = \sum_k P(k) \theta^k$$

Finding S



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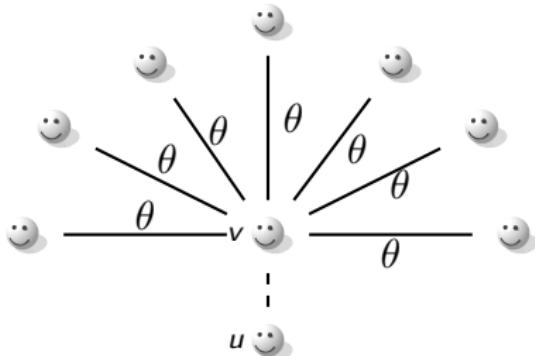
Probability a random **degree- k** test individual remains susceptible is

$$S = \sum_k P(k) \theta^k = \psi(\theta)$$

where

$$\psi(x) = \sum_k P(k) x^k$$

Finding θ

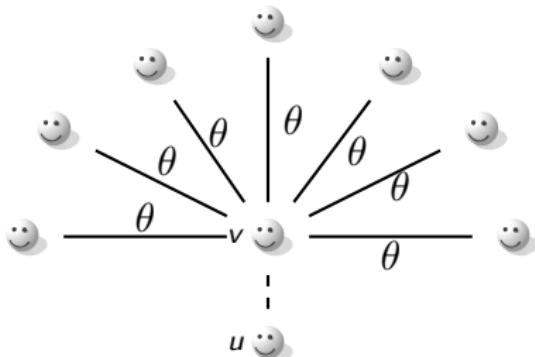


Probability a random degree k partner still susceptible is

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Finding θ

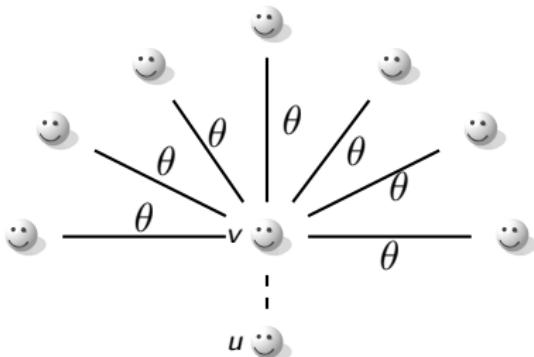


Probability a random ~~degree- k~~ partner still susceptible is

$$\phi_S = \sum_k P_n(k) \theta^{k-1}$$



Finding θ

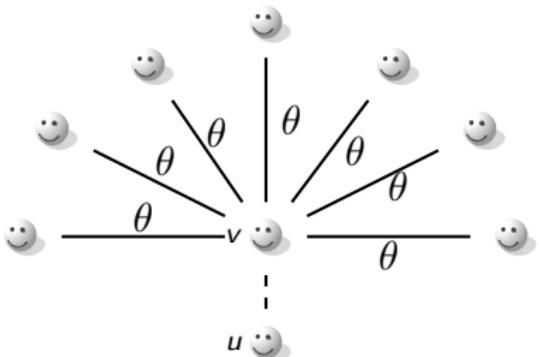


Probability a random ~~degree- k~~ partner still susceptible is

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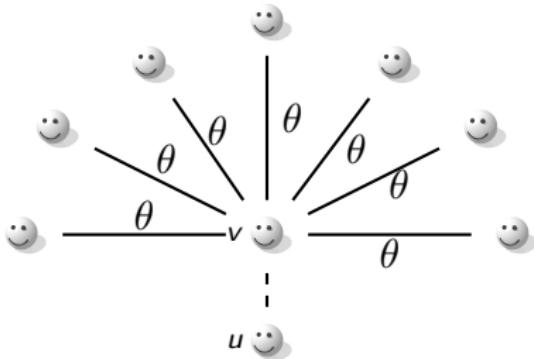
Finding θ



Probability a random ~~degree- k~~ partner still susceptible is

$$\phi_S = \sum_k \frac{kP(k)}{\langle K \rangle} \theta^{k-1} = \frac{\psi'(\theta)}{\psi'(1)}$$

Finding θ



Probability a random ~~degree-k~~ partner still susceptible is

$$\phi_S = \sum_k \frac{kP(k)}{\langle K \rangle} \theta^{k-1} = \frac{\psi'(\theta)}{\psi'(1)}$$

If $T = \beta/(\beta + \gamma)$, then probability partner does not transmit to u is

$$\theta = \phi_S + (1 - T)(1 - \phi_S) = 1 - T + T \frac{\psi'(\theta)}{\psi'(1)}$$

Final Size

So

$$R = 1 - \psi(\theta)$$

where

$$\theta = 1 - T + T \frac{\psi'(\theta)}{\psi'(1)}$$



SIR epidemics in Static Networks

More Contact Structure

Epidemic Probability

Final Size

Dynamics



Our derivation here follows [11]. This is not the only approach — It has significantly fewer equations than other approaches [63, 7, 64, 65], but the predictions of the models are identical (subject to some small caveats)

Calculating Dynamics

The network structure alters the infection process (but not the recoveries)

Calculating Dynamics

The network structure alters the infection process (but not the recoveries)



$$I = 1 - S - R, \quad \dot{R} = \gamma I$$

We will switch to a partnership-based perspective to find $S(t)$.

Revisiting the test individual

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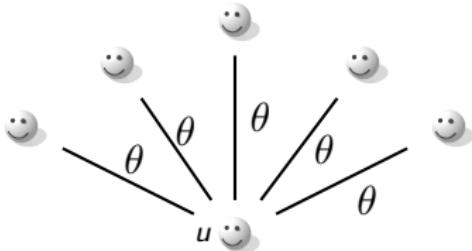
$$S(t) = P(u \text{ is susceptible})$$

- Let v be a random partner of u .
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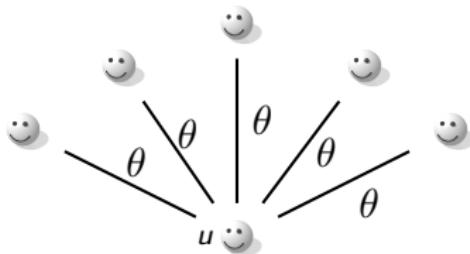
Finding $S(t)$



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Finding $S(t)$



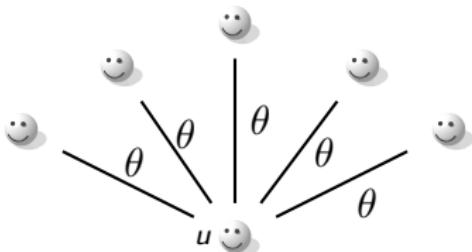
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Finding $S(t)$



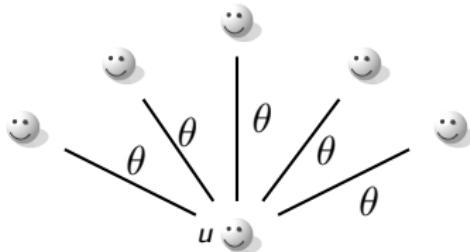
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Probability a random **degree- k** test individual still susceptible is

$$S(t) = \sum_k P(k) \theta(t)^k$$



Finding $S(t)$



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Probability a random **degree- k** test individual still susceptible is

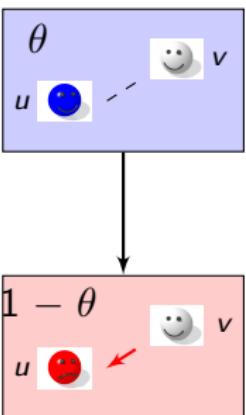
$$S(t) = \sum_k P(k) \theta(t)^k = \psi(\theta(t))$$

where

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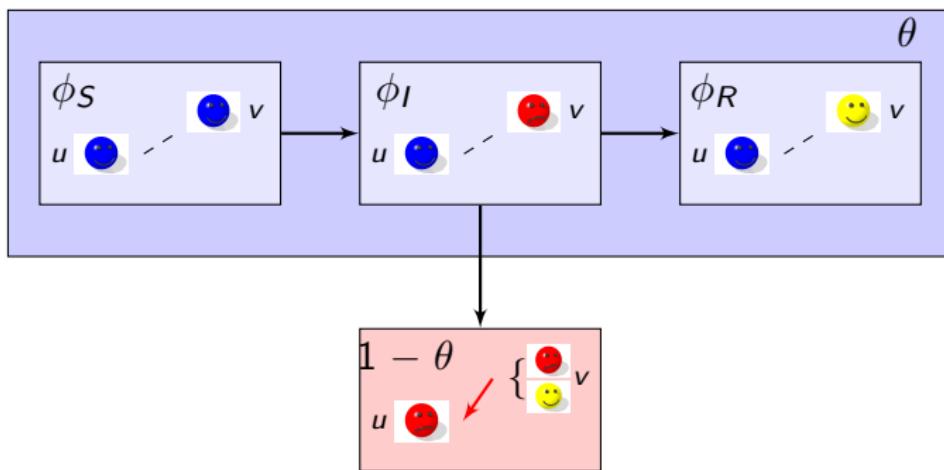


How does θ evolve?



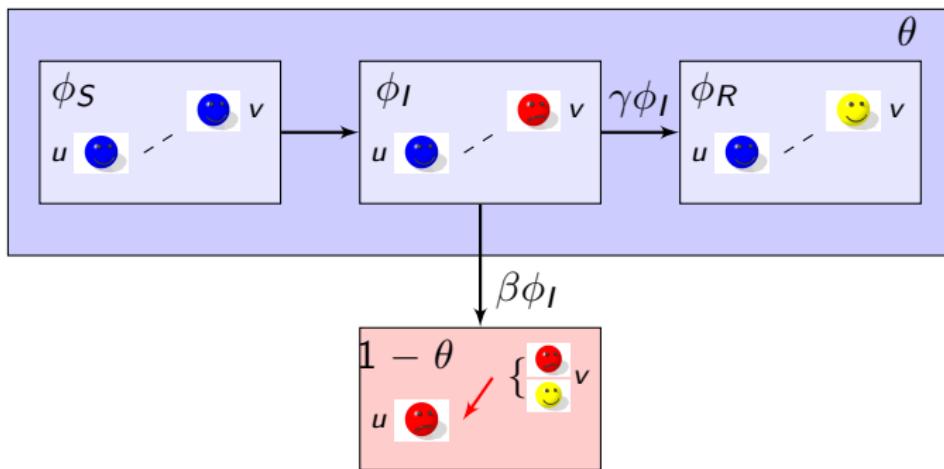


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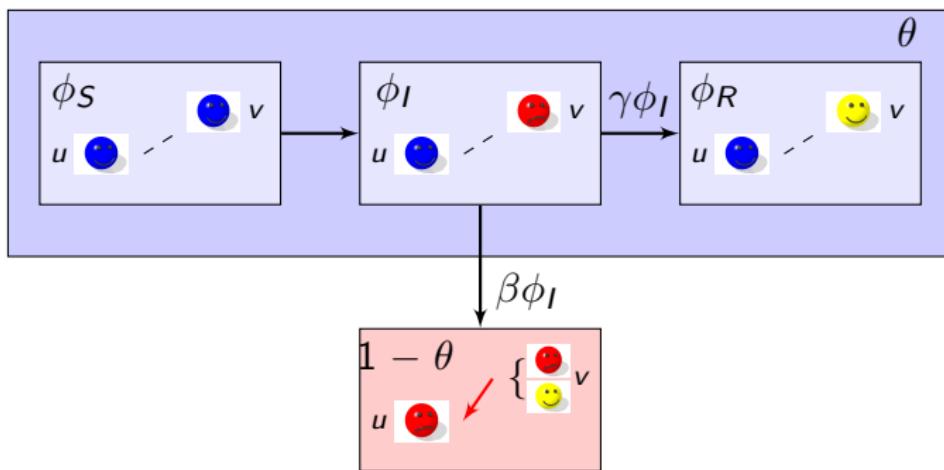
- $\theta = \phi_S + \phi_I + \phi_R.$

How does θ evolve?



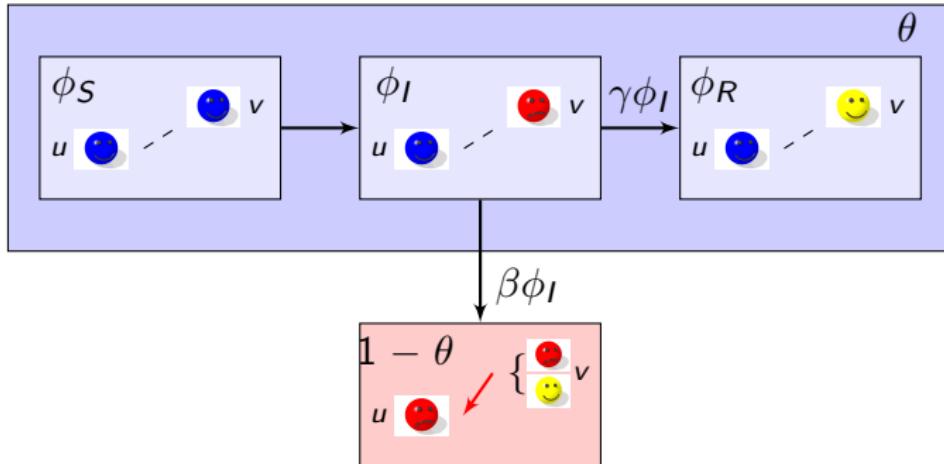
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- $\dot{\theta} = -\beta\phi_I$.

How does θ evolve?



- $\theta = \phi_S + \phi_I + \phi_R$.
- $\dot{\theta} = -\beta\phi_I$.
- Our goal is to find ϕ_I in terms of θ .

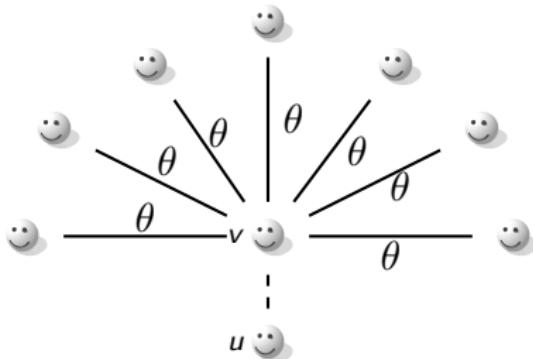
Finding $\phi_R(t)$



Because derivatives are proportional, $\phi_R = \frac{\gamma}{\beta}(1 - \theta)$



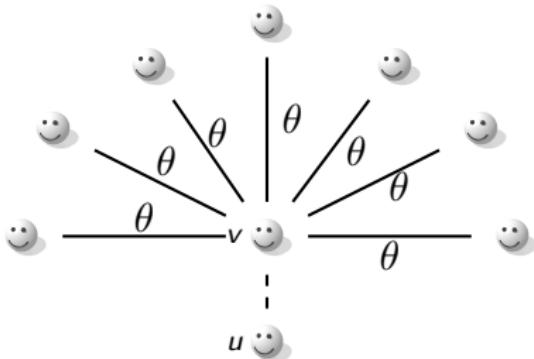
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Finding $\phi_S(t)$

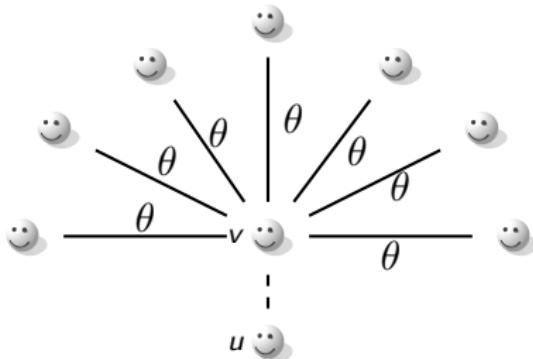


Probability a random **degree- k** partner still susceptible is

$$\phi_S(t) = \sum_k P_n(k) \theta(t)^{k-1}$$



Finding $\phi_S(t)$

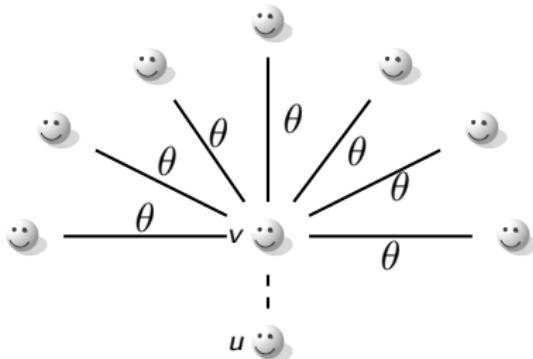


Probability a random **degree- k** partner still susceptible is

$$\phi_S(t) = \sum_k \frac{kP(k)}{\langle K \rangle} \theta(t)^{k-1}$$

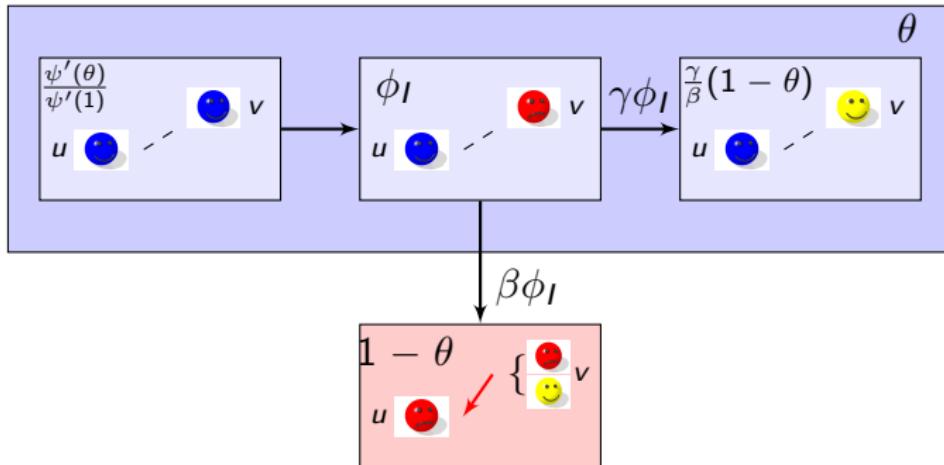


Finding $\phi_S(t)$

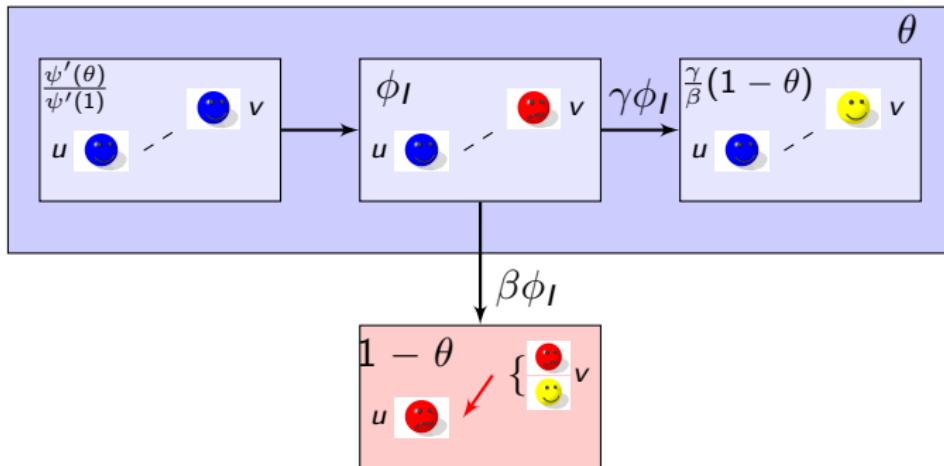


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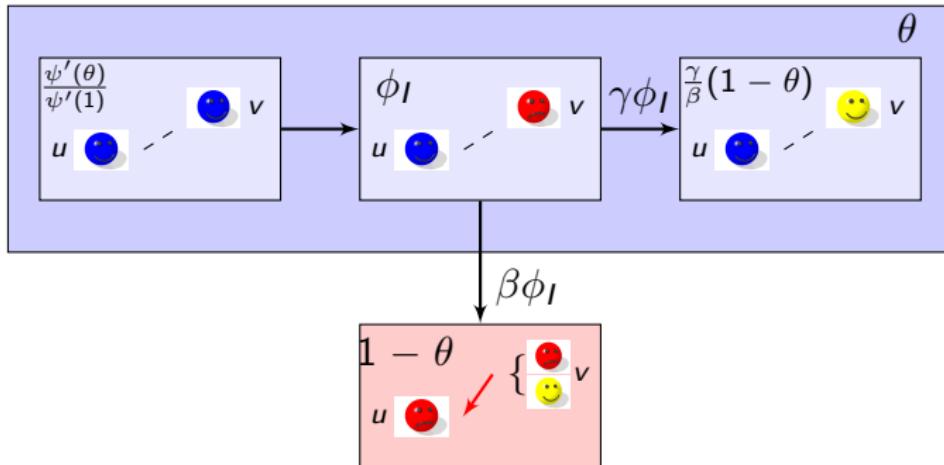
$$\phi_S(t) = \sum_k \frac{kP(k)}{\langle K \rangle} \theta(t)^{k-1} = \frac{\psi'(\theta)}{\psi'(1)}$$



Since $\phi_I = \theta - \phi_S - \phi_R$



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Since $\phi_I = \theta - \phi_S - \phi_R = \theta - \frac{\psi'(\theta)}{\psi'(1)} - \frac{\gamma}{\beta}(1 - \theta)$, we have

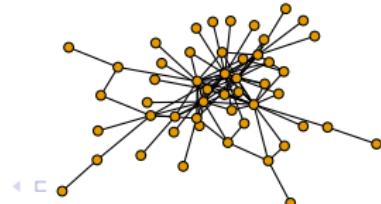
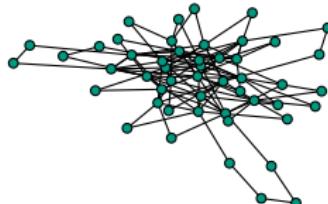
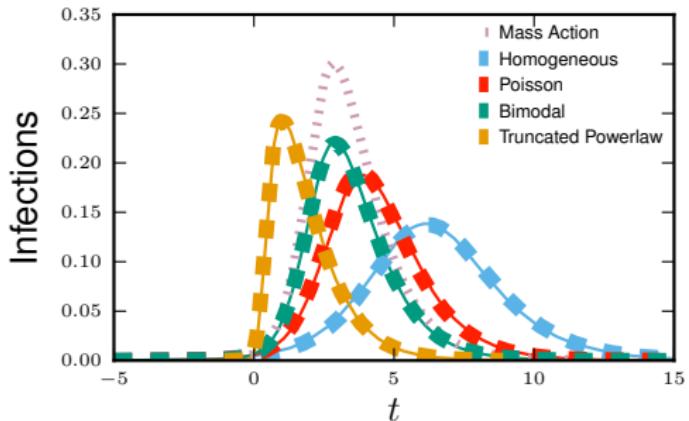
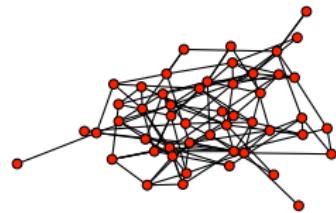
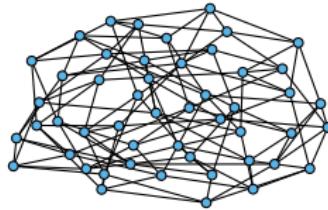
$$\dot{\theta} = -\beta\phi_I = -\beta\theta + \beta \frac{\psi'(\theta)}{\psi'(1)} + \gamma(1 - \theta)$$

Final System

We finally have

$$\dot{\theta} = -\beta\theta + \beta \frac{\psi'(\theta)}{\psi'(1)} + \gamma(1 - \theta)$$

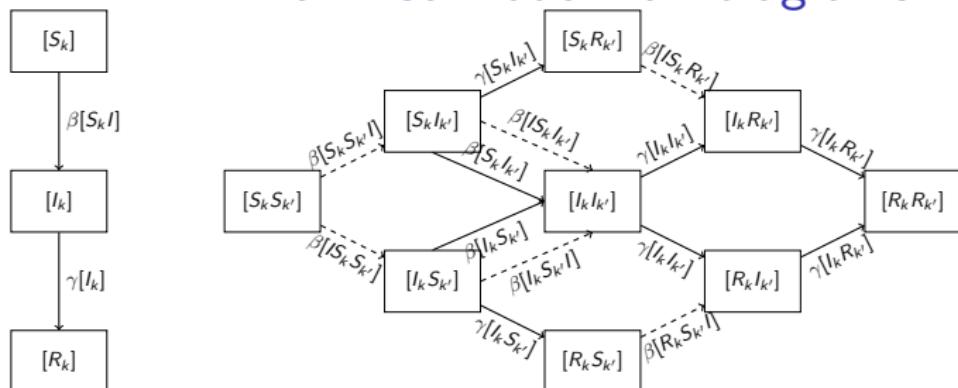
$$\dot{R} = \gamma I \quad S = \psi(\theta) \quad I = 1 - S - R$$



Other formulations

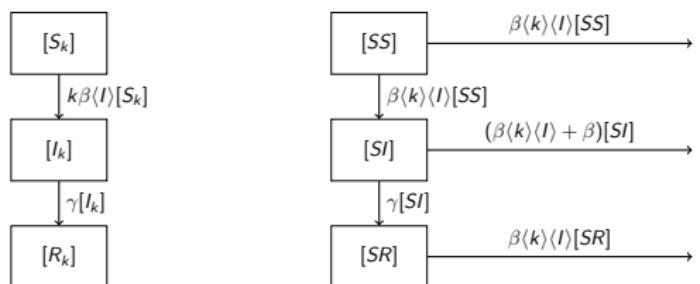
- Other variable choices exist, which lead to different equations.
- Subject to mild conditions the equations are all equivalent [66].
- The number of equations and compartments can vary substantially

Pairwise model flow diagrams



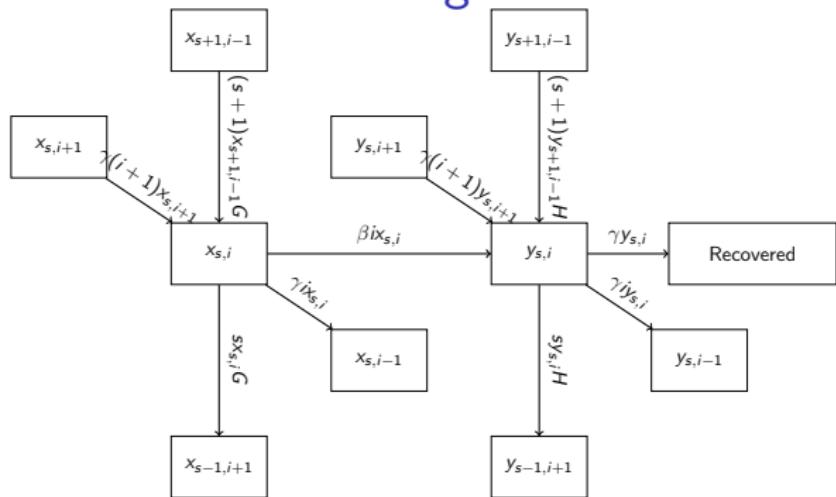
- The flow diagram underlying the basic pairwise model [7].
- We track individuals of each status and degree as well as partnerships between individuals of various statuses and degrees.
- Dashed lines denote transitions that rely on infection coming from a source outside the edge of interest.
- The triples $[A_k' S_k I]$ and $[IS_k A_k']$ can be expressed in terms of the doubles and singles: $[A_k' S_k][S_k I]/[S_k]$

Reduced pairwise flow diagram



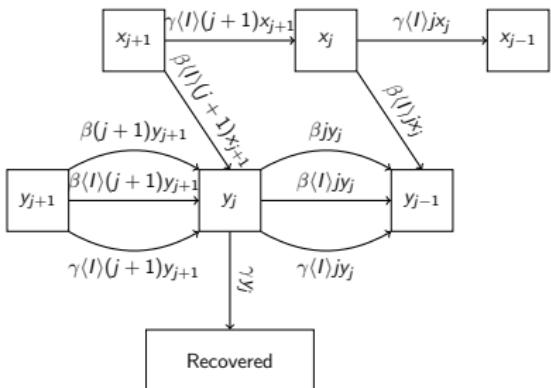
- The flow diagram for the reduced system of [64].
- The $[SS]$, $[SI]$, and $[SR]$ compartments correspond to the sum of the $[S_k S_{k'}]$, $[S_k I_{k'}]$ and $[S_k R_{k'}]$ compartments of the basic pairwise model.
- $\langle I \rangle = [SI]/([SS] + [SI] + [SR])$

Effective degree model flow diagram

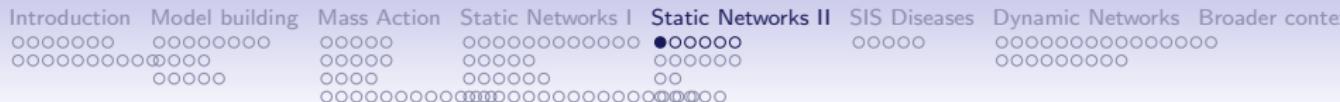


- The flow diagram underlying the effective degree model of [63].
- We include just the fluxes involving the $x_{s,i}$ or $y_{s,i}$ compartments. Fluxes between other compartments exist but are not included.

Compact effective degree model flow diagram



- The flow diagram that underlies the model of [65].
- Only the fluxes into and out of y_j and x_j are included. Fluxes between other compartments exist, but are not included.
- An active edge is eliminated if the partner recovers or if it transmits infection in either direction. The quantity $\langle I \rangle$ represents the probability an active edge joins an individual with an infected partner.



Static Networks II

\mathcal{R}_0

Clustered networks

Heterogeneous infectiousness/susceptibility

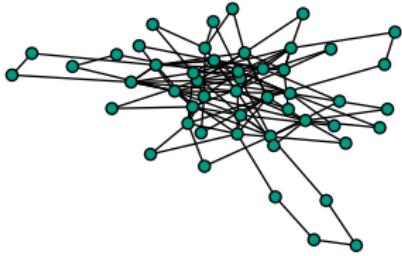
Multiple Diseases

Epidemic Thresholds

- Consider a population in which $P(2) = 0.5$ and $P(8) = 0.5$.

Epidemic Thresholds

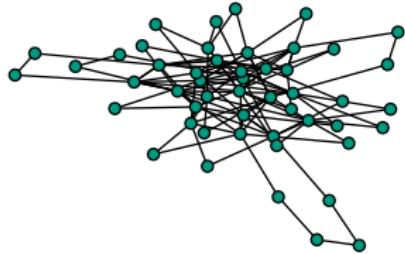
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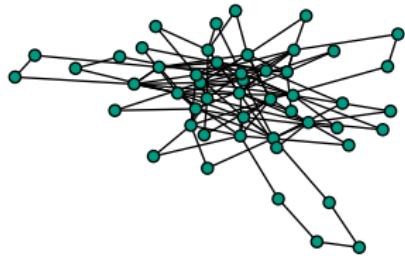


- The transmission probability is $T = \beta / (\beta + \gamma)$.



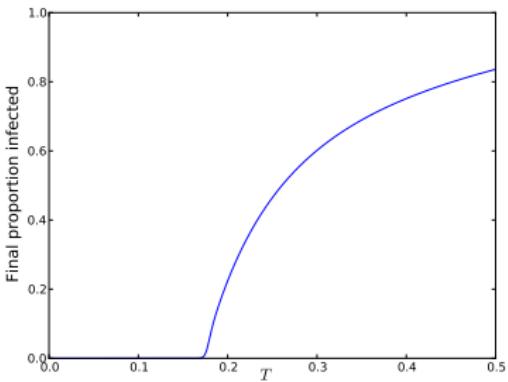
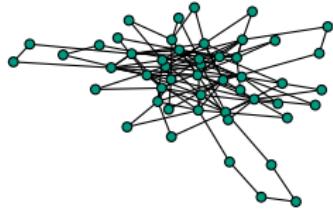
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- The transmission probability is $T = \beta / (\beta + \gamma)$.
- Can you guess what threshold value of T is needed for an epidemic?

Epidemic Thresholds



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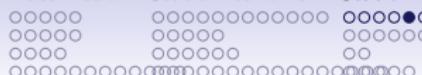
$$\mathcal{R}_0$$

- We define \mathcal{R}_0 as the average number of new infections caused by an infected individual early in an outbreak.
- Note that this definition does not look at the number of cases caused by an average individual introduced to the population.
- If $\mathcal{R}_0 > 1$ then epidemics are possible (but not guaranteed). If $\mathcal{R}_0 < 1$, they are impossible.



$$\mathcal{R}_0$$

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- So the average infected individual infects
$$\mathcal{R}_0 = \sum_k P_n(k)(k - 1)T$$



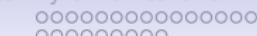
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- For a given average degree, larger variance gives larger \mathcal{R}_0 .
- If $P(2) = P(8) = 0.5$, then $\mathcal{R}_0 = 5.8T$.
- So $T = 1/5.8$ is the epidemic threshold.

Review of Configuration Model networks

For static configuration model networks, we have

- Calculated the probability of an epidemic (by calculating the probability no 'daughter' of the index case leads to an epidemic)
- Calculated the final size of an epidemic (by calculating the probability a random individual is never infected)
- Calculated the dynamics of an epidemic (by calculating the probability a random individual is not infected by time t).
- Calculated \mathcal{R}_0 (by calculating the expected number of transmissions caused by an early - nonindex - case).

In all cases we had to account for size bias: A random individual's partner has higher expected degree than a random individual.



Static Networks II

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Multiple Diseases

Epidemics in clustered networks

- A clustered network is thought of as a network with many triangles in it.

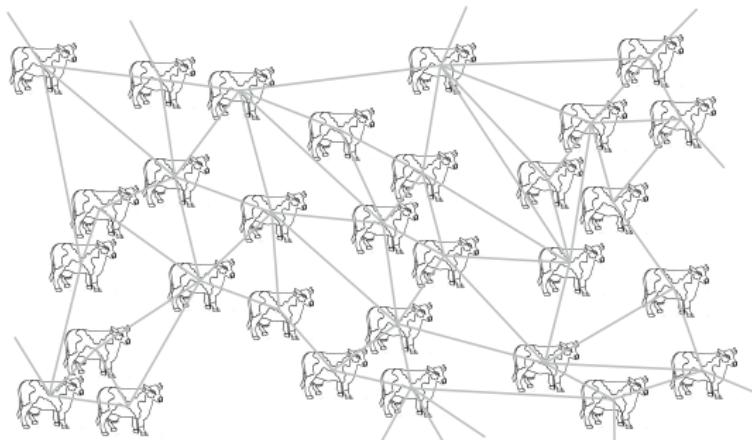


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- More generally, for our purposes a clustered network is any network with enough short cycles to affect disease transmission.

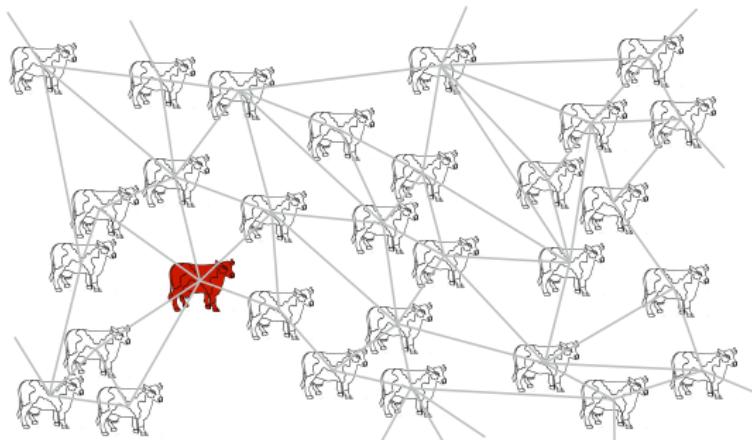
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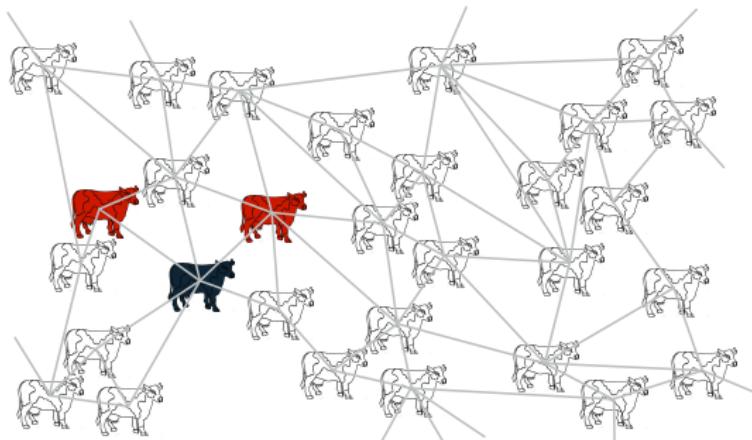
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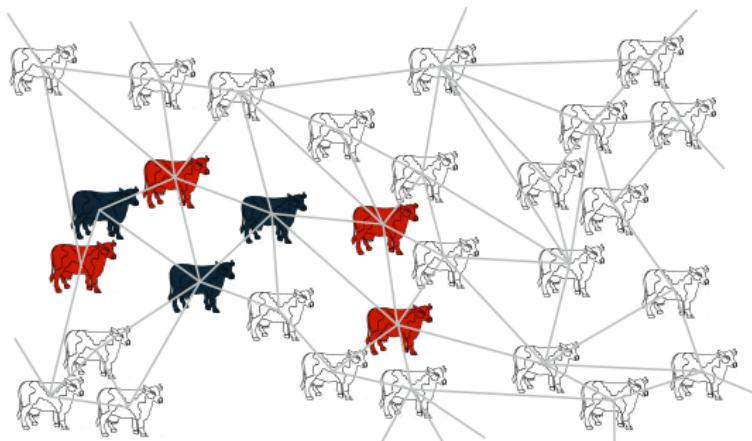
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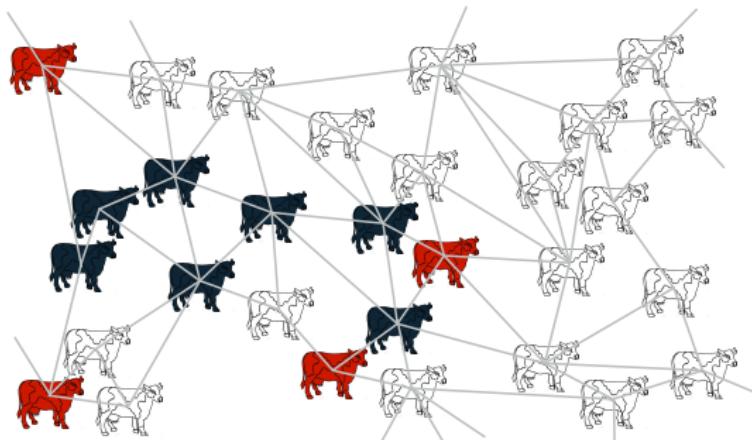
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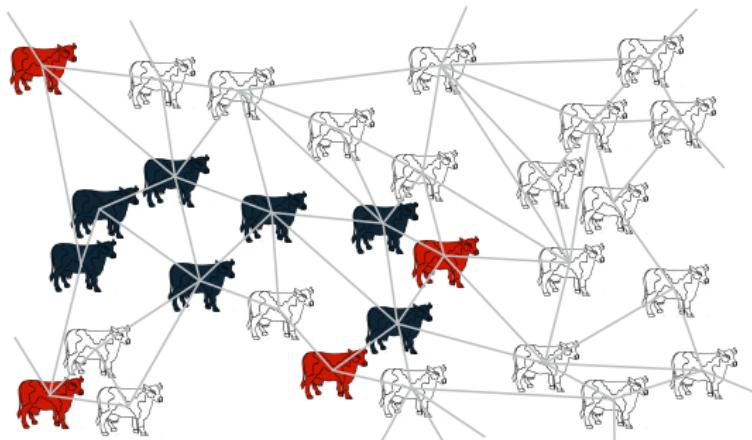
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Density of triangles can be measured as “transitivity” or “clustering”.

Epidemics in clustered networks

- The difficulty in modeling disease spread comes from the fact that we cannot treat partners as independent: a formula like $S = \sum_k P(k)\theta^k$ fails because transmissions are not independent.

Epidemics in clustered networks

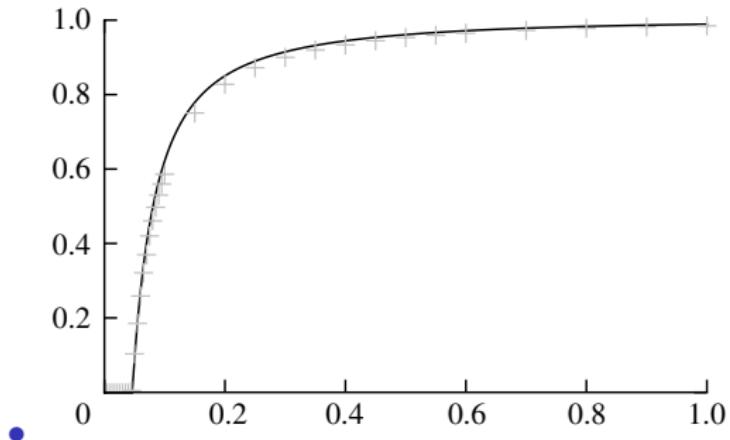
- The difficulty in modeling disease spread comes from the fact that we cannot treat partners as independent: a formula like $S = \sum_k P(k)\theta^k$ fails because transmissions are not independent.
- There are some workarounds. . .

workarounds for clustered networks

- If transmission probability is low, and degrees are large, then it is not a bad approximation to say that the statuses of two partners are independent [36]

workarounds for clustered networks

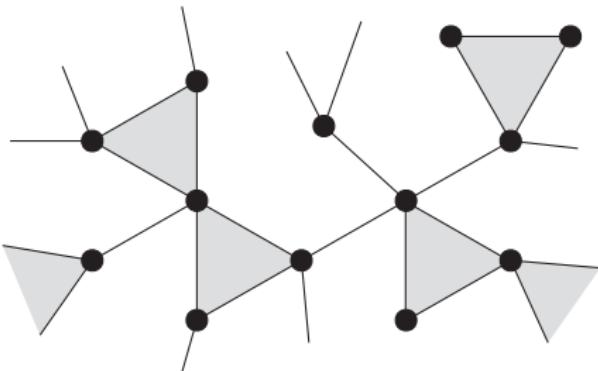
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- Horizontal axis is transmission probability, vertical axis is epidemic size. Solid curve is final size prediction ignoring clustering, symbols are simulation.

workarounds for clustered networks

- In a very special case we can recover some sort of independence. We can generate random graphs containing triangles for which no two triangles share edges. Then each triangle can be thought of as independent of any others.

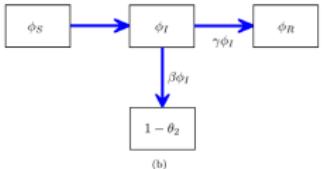
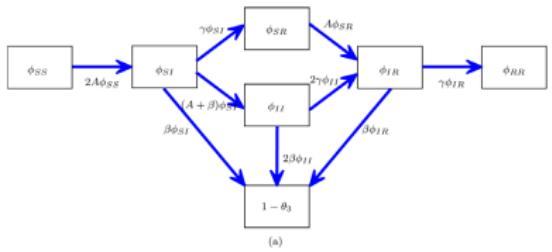


workarounds for clustered networks

- We can draw flow diagrams much as before, but need separate diagrams for triangles and other edges.

workarounds for clustered networks

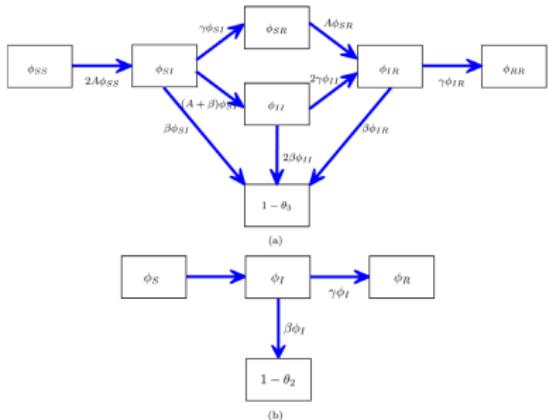
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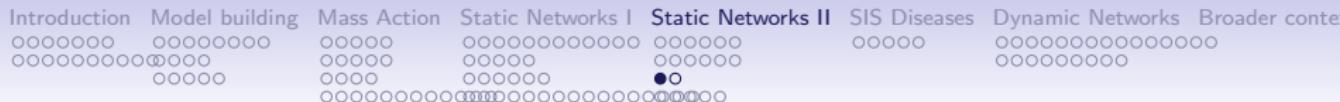
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workarounds for clustered networks

- We can draw flow diagrams much as before, but need separate diagrams for triangles and other edges.



- This requires many more equations.



Static Networks II

\mathcal{R}_0

Clustered networks

Heterogeneous infectiousness/susceptibility

Multiple Diseases

Impact of heterogeneous infectiousness/susceptibility

- We can derive results about the impact of heterogeneity in infectiousness and/or susceptibility
- For a given average transmission probability, the probability of an epidemic is reduced if there is heterogeneity in infectiousness.
- For a given average transmission probability, the size of an epidemic is reduced if there is heterogeneity in susceptibility.
- These results hold regardless of whether the network is clustered.



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Multiple Diseases

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- Having one disease may increase or reduce susceptibility and transmissibility for the other disease.
- Examples: two related strains of influenza; HIV and Herpes.
- It is difficult to disentangle increased susceptibility from simply being more likely to have an infected partner (HIV/Herpes).
- Would treating Herpes be an effective HIV intervention?

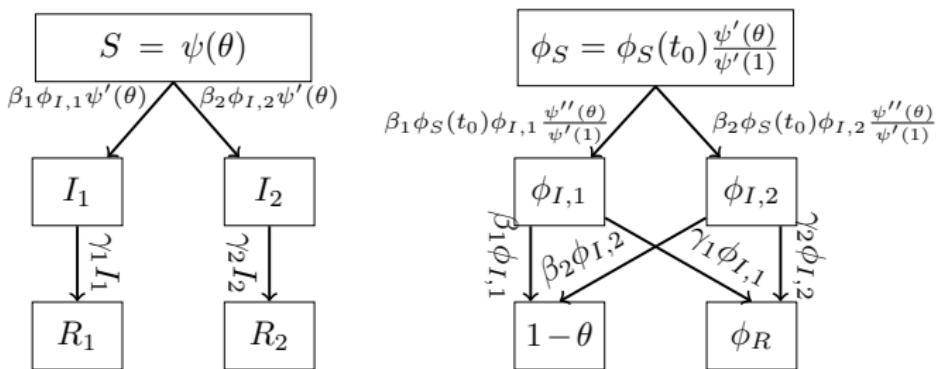
Mutually exclusive diseases

- If having one disease gives complete immunity to the other disease(s), we can derive equations for a configuration model network. [64, 67]

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Mutually exclusive diseases

- The equations become

$$\dot{\theta} = -\beta_1 \phi_{I,1} - \beta_2 \phi_{I,2}$$

$$\dot{\phi}_{I,m} = -(\beta_m + \gamma_m) \phi_{I,m} + \beta_m \phi_{I,m} \phi_S(t_0) \frac{\psi''(\theta)}{\psi'(1)}$$

$$S = \psi(\theta)$$

$$\dot{I}_m = \beta_m \phi_{I,m} \psi'(\theta) - \gamma_m I_m$$

$$\dot{R}_m = \gamma_m I_m$$

- For more detail, see [67] for full dynamics of interacting diseases and [68, 69] for final sizes (assuming one disease spreads before the other).



Non-exclusive diseases

- I'm not familiar with simulation work for diseases that aren't exclusive (I'm sure it exists).
- In [70], the authors considered one disease spreading and (after the epidemic) a second disease spreads only on those individuals infected by the first. Final size results only.
- I have a paper in progress which deals with the full dynamics. It's pretty complicated.



SIS Diseases

SIS

SIS Disease

SIS diseases appear to be hard.

- S is no longer the probability of having escaped every infection.
- We cannot treat partners of a given individual as independent of one another: an individual who has been infected and recovered is likely to have more infected partners than an individual who has not been infected.

Dynamic networks

- If partnerships are very brief, then many of the problems go away.
- In this limit we can write down exact equations, but they are not as compact ([10] and many others)

$$\dot{S}_1 = \gamma I_1 - \langle I \rangle S_1$$

$$\dot{I}_1 = -\gamma I_1 + \langle I \rangle S_1$$

...

$$\dot{S}_k = \gamma I_k - k \langle I \rangle S_k$$

$$\dot{I}_k = -\gamma I_k + k \langle I \rangle S_k$$

...

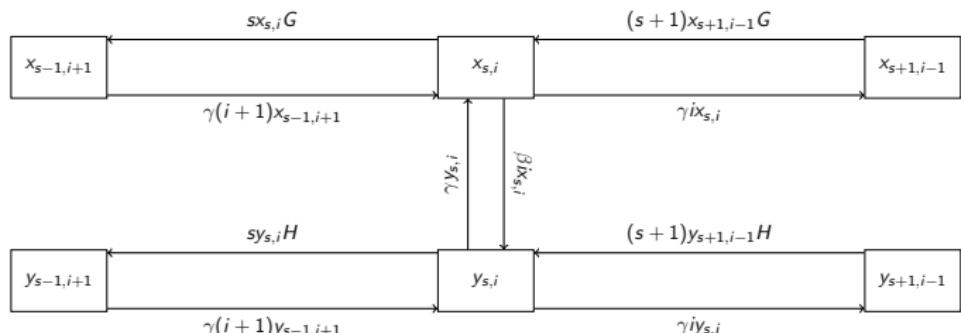
where $\langle I \rangle$ is the probability a partner is infected:

$$\langle I \rangle = \sum_k k I_k / \sum_k k (I_k + S_k).$$

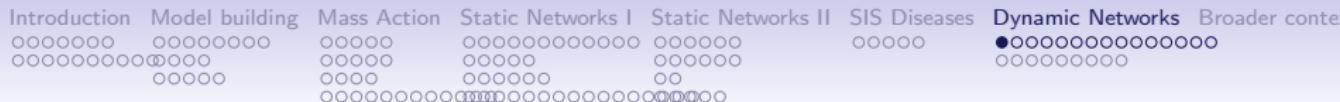
Non-zero partnership duration

- If partnerships are permanent, then some other approach is needed.
- The main approach used here does not appear easy to adapt.
- Some of the pair-based methods have been used, with varying success. See for example [71, 63].
- Many of the general network effects in the introduction remain true.

The model of Lindquist et al [63]



- $x_{s,i}$ is the number of susceptible individuals with s susceptible and i infected partners
- $y_{s,i}$ is the number of infected individuals with s susceptible and i infected partners.
- $H = \beta \frac{\sum_{k=1}^M \sum_{j+l=k} l^2 S_{jl}}{\sum_{k=1}^M \sum_{j+l=k} jl j_l}$ and $G = \beta \frac{\sum_{k=1}^M \sum_{j+l=k} jl x_{jl}}{\sum_{k=1}^M \sum_{j+l=k} j x_{jl}}$



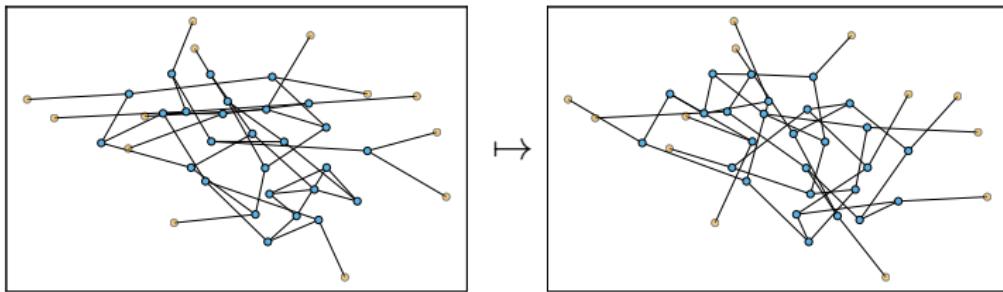
SIR Epidemics in Dynamic Networks short duration partnerships Dormant Contact model

Dynamic Networks with short partnership duration

- We now drop the assumption that the partnerships are permanent.
- But we make the opposite assumption that partnerships are fleeting (very, very brief).
- We can still think of k as the number of partners an individual has, but we assume those partners change from one moment to the next. Its value varies from individual to individual.
- This model is appropriate if partnerships are short enough that we can neglect multiple transmissions in the same partnership.



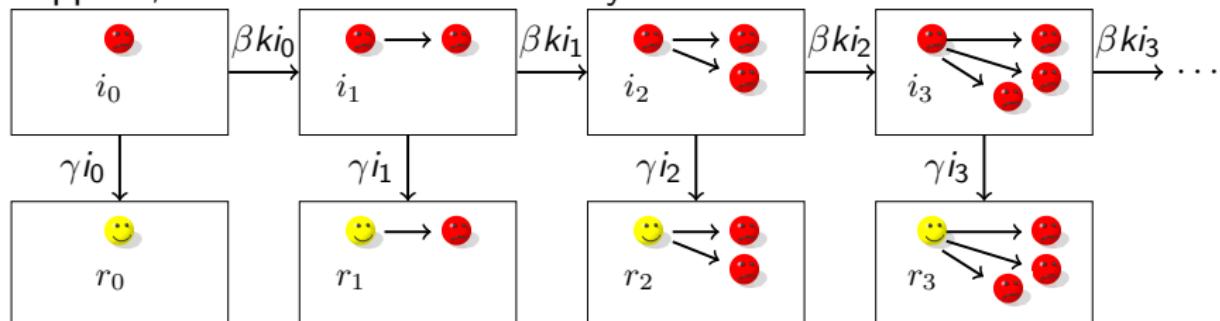
Different times for same population



The degree of every individual remains the same, but the partners are changed.

Calculating \mathcal{P} .

We follow the Mass Action approach. When a transmission happens, that stub can immediately infect another individual.



Consider an individual u who becomes infected at time $t = 0$. Let $r(m|k)$ be the probability of causing m transmissions given k stubs. Following steps before, we have

$$r(m|k) = \left(\frac{k\beta}{k\beta + \gamma} \right)^m \frac{\gamma}{k\beta + \gamma}$$

typo & notation change

Finding \mathcal{P}

- The probability of not causing an epidemic is

$$1 - \mathcal{P} = \sum_{k=0}^{\infty} \left(P(k) \sum_{m=0}^{\infty} r(m|k) \alpha^m \right)$$

where α is the probability that a secondary case does not cause epidemics.

- After some work, we can show that

$$\sum_{m=0}^{\infty} r(m|k) \alpha^m = \frac{\gamma}{k\beta + \gamma} \sum_{m=0}^{\infty} \left(\frac{k\beta}{k\beta + \gamma} \right)^m \alpha^m = \frac{\gamma}{k\beta(1 - \alpha) + \gamma}$$

- To find α , we note that

$$\alpha = \sum_{k=0}^{\infty} P_n(k) \sum_{m=0}^{\infty} r(m|k) \alpha^m = \sum_{k=0}^{\infty} \frac{kP(k)}{\langle K \rangle} \frac{\gamma}{k\beta(1 - \alpha) + \gamma}$$

Finding \mathcal{P}

So we have

$$1 - \mathcal{P} = \sum_{k=0}^{\infty} P(k) \frac{\gamma}{k\beta(1 - \alpha) + \gamma}$$

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This can be solved numerically.

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- To find ξ , we sum over all k the probability a stub belongs to an individual of degree k times the expected number of transmission caused. thought question: why $kP(k)/\langle K \rangle$ rather than $P(k)$?

$$\xi = \sum_{k=0}^{\infty} \frac{kP(k)}{\langle K \rangle} (1 - \theta^k) \beta / \gamma$$

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$$\begin{aligned}
 \xi &= \sum_{k=0}^{\infty} \frac{kP(k)}{\langle K \rangle} (1 - \theta^k) \beta / \gamma = \frac{\beta}{\gamma} \left(\sum \frac{kP(k)}{\langle K \rangle} - \sum \theta \frac{kP(k)\theta^{k-1}}{\langle K \rangle} \right) \\
 &= \frac{\beta}{\gamma} \left(1 - \theta \frac{\psi'(\theta)}{\psi'(1)} \right)
 \end{aligned}$$

Final Size

So

$$\theta = e^{-\xi}$$

$$\xi = \frac{\beta}{\gamma} \left(1 - \theta \frac{\psi'(\theta)}{\psi'(1)} \right)$$

gives θ and ξ . The final size is $1 - \psi(\theta)$.

Calculating dynamics

We must generalize our definitions of ϕ_S , ϕ_I , and ϕ_R . As before, u is a test individual.

- ϕ_S : the probability that a stub belonging to u has not carried infection to u and is currently connected to a susceptible individual.
- ϕ_I : as above, but connected to an infected individual.
- ϕ_R : as above, but connected to a recovered individual.

Calculating dynamics

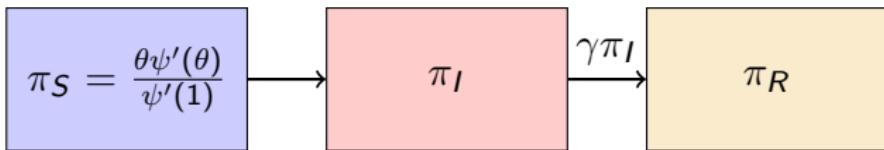
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- Since edges are reforming at all times, the probability of connecting to a susceptible individual is proportional to the number of stubs susceptible individuals have.

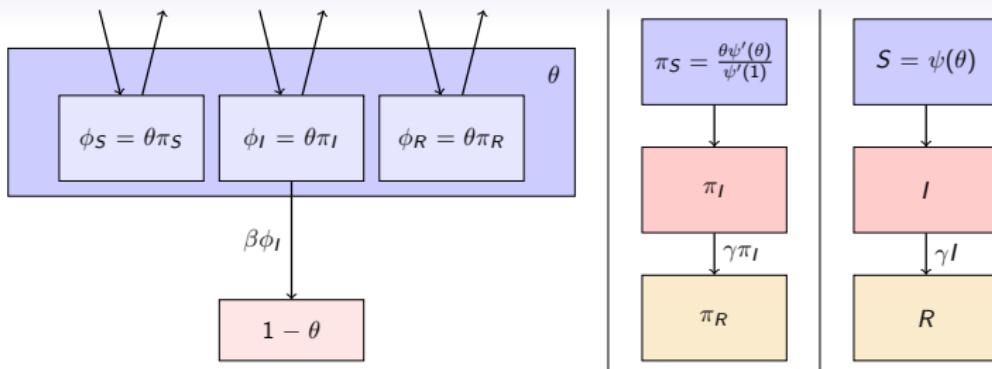
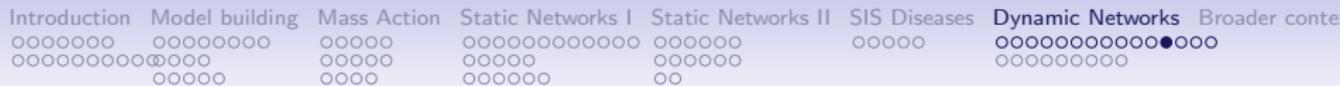
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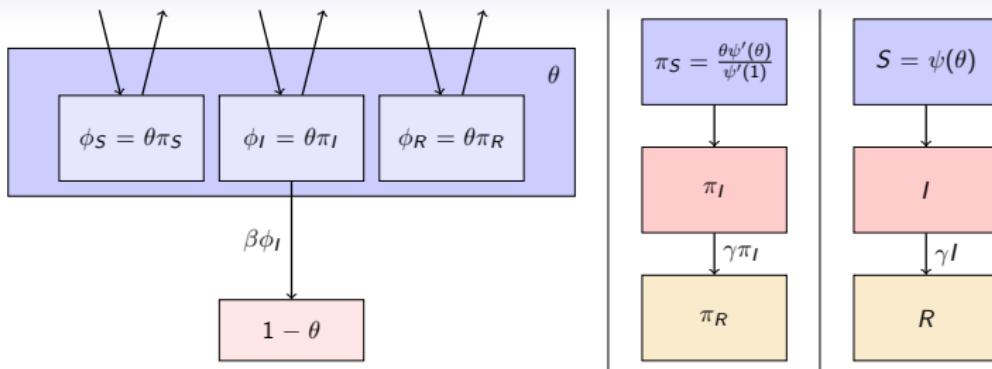
- We must calculate the probability that an edge connects to an individual of each type.
- Since edges are reforming at all times, the probability of connecting to a susceptible individual is proportional to the number of stubs susceptible individuals have.
- So we must calculate the proportion of stubs belonging to individuals of each type.



We define

- π_S to be the probability a stub belongs to a susceptible individual. We have $\pi_S = \sum_k \frac{kP(k)}{\langle K \rangle} \theta^k = \theta\psi'(\theta)/\psi'(1)$
- π_I to be the probability a stub belongs to an infected individual. $\pi_I = 1 - \pi_S - \pi_R$
- π_R to be the probability a stub belongs to a recovered individual. $\dot{\pi}_R = \gamma\pi_I$

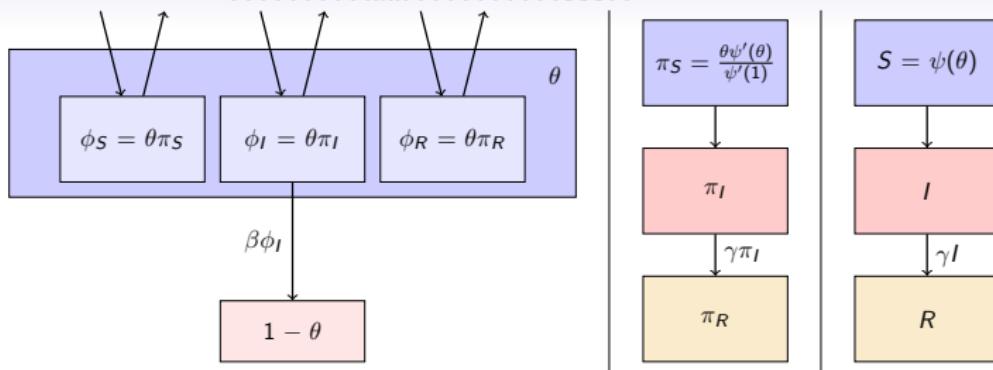




- We have

$$\dot{\pi}_R = \gamma\pi_I = \gamma\phi_I/\theta = -(\gamma/\beta)\dot{\theta}/\theta$$

$$\text{So } \pi_R = -(\gamma/\beta) \ln \theta$$

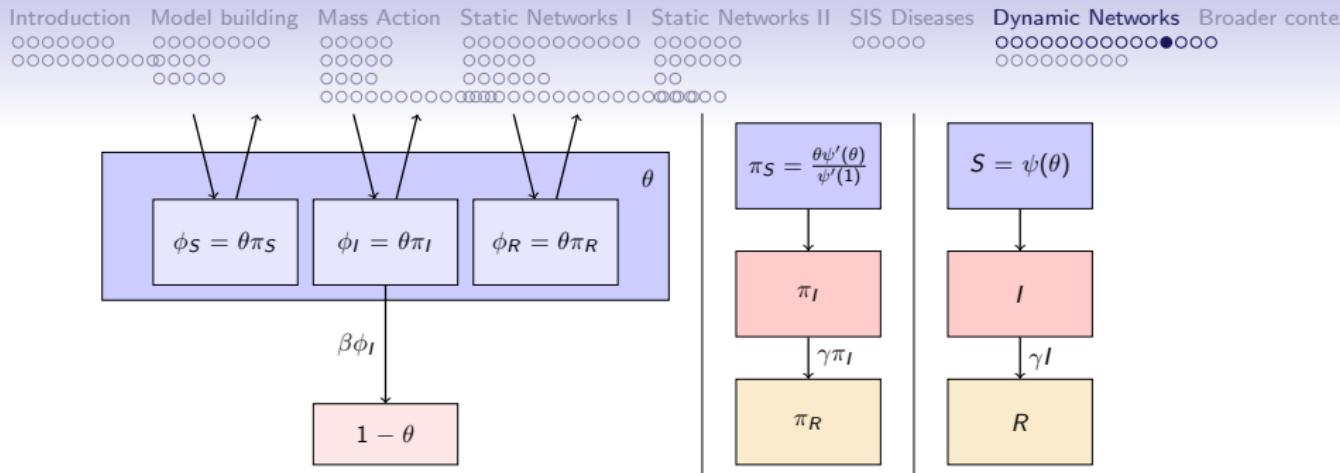


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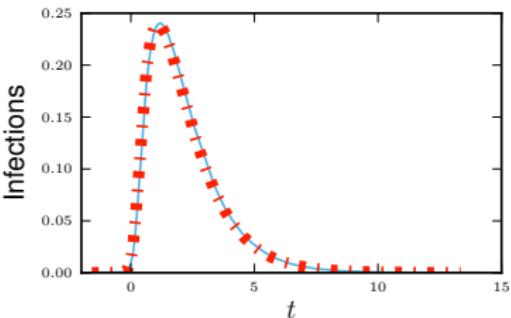
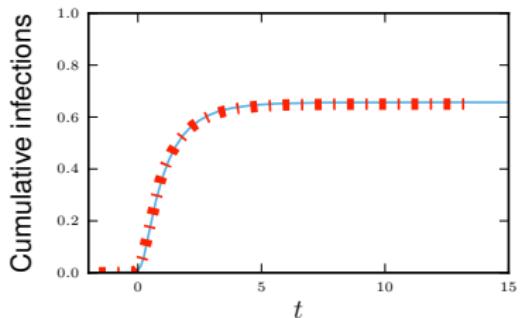
$$\bullet \quad \pi_I = 1 - \frac{\theta\psi'(\theta)}{\psi'(1)} + \frac{\gamma}{\beta}\ln\theta$$

- So since $\dot{\theta} = -\beta\phi_I = -\beta\theta\pi_I$ we get

$$\dot{\theta} = -\beta\theta + \beta\frac{\theta^2\psi'(\theta)}{\psi'(1)} - \theta\gamma\ln\theta,$$

$$\dot{R} = \gamma I, \quad S = \psi(\theta), \quad I = 1 - S - R.$$

Comparison with simulation



Epidemics in a network of fleeting partnerships with $P(1) = 25/31$, $P(5) = 5/31$, and $P(25) = 1/31$ and $\beta = \gamma = 1$.

Final size revisited

- Our equations were

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then $-\gamma\xi - \gamma \ln \theta = 0$.

- So we conclude that

$$\theta = e^{-\xi}$$

\mathcal{R}_0 and thresholds

- Early in an epidemic, an average infected individual has degree k with probability $P_n(k) = kP(k)/\langle K \rangle$.

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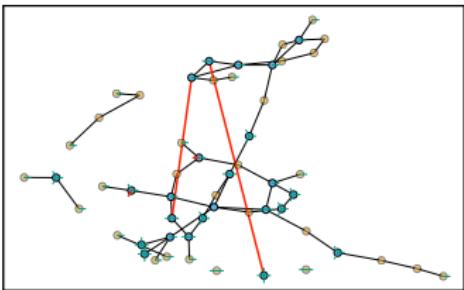
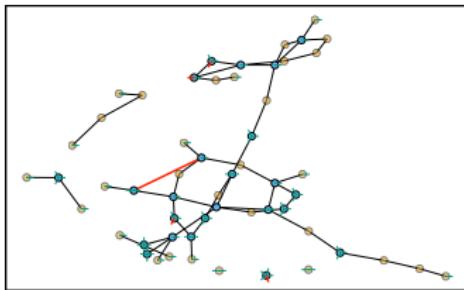
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We can also derive this by careful examination of $\sum_{m=0}^{\infty} mr(m|k)$.
- So

$$\mathcal{R}_0 = \sum_{k=0}^{\infty} \frac{kP(k)}{\langle K \rangle} k \frac{\beta}{\gamma} = \frac{\beta \langle K^2 \rangle}{\gamma \langle K \rangle}$$

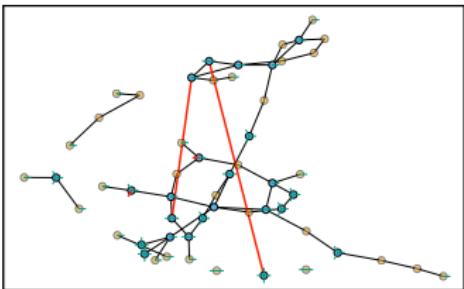
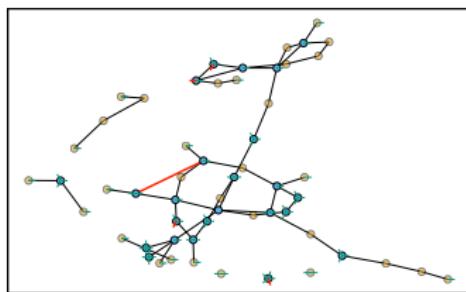
SIR Epidemics in Dynamic Networks

short duration partnerships
Dormant Contact model

A general network model

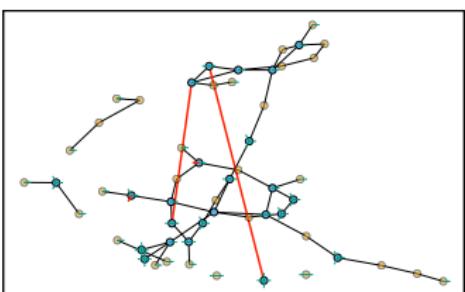
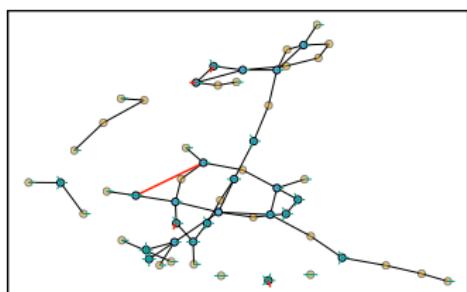


A general network model



- We can think of each individual as having some number of stubs k .

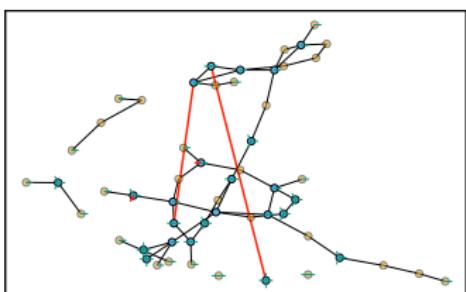
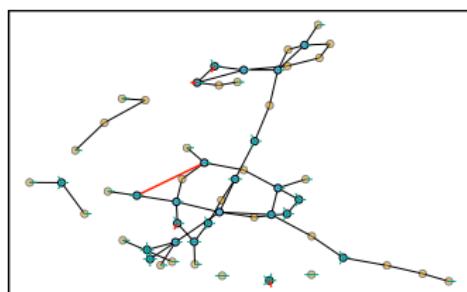
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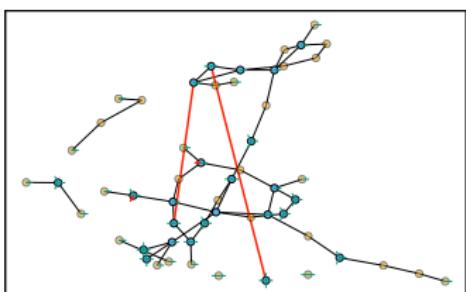
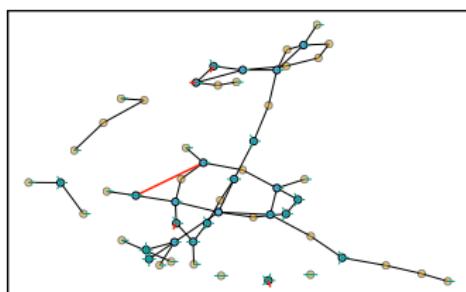


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- Active stubs become dormant at rate η_2 .

edge dynamics

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edge dynamics

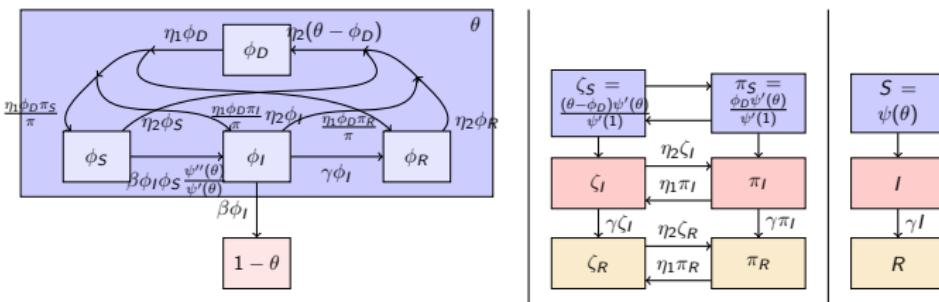
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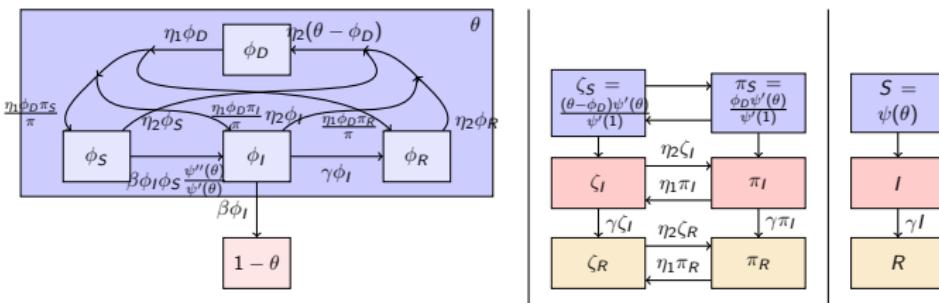
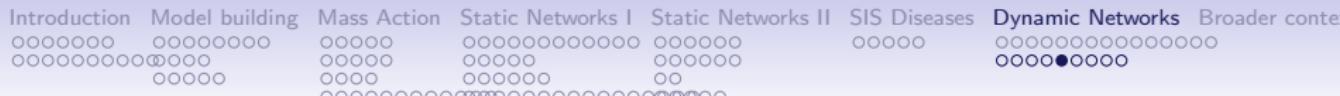
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 - Every stub is part of one of these:
- $$1 = \pi_S + \pi_I + \pi_R + \zeta_S + \zeta_I + \zeta_R.$$

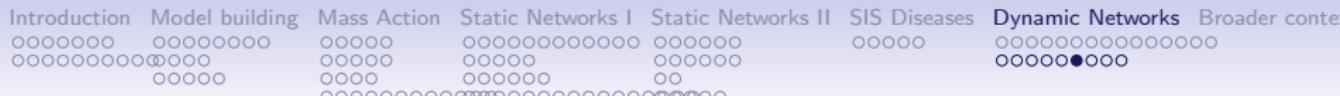
We end up with



- We define ϕ_S to be the probability a stub is active, connected to a susceptible individual, and has never transmitted infection to the given random individual. Similarly ϕ_I , and ϕ_R .
- We similarly define ϕ_D to be the probability the stub is dormant and has never transmitted infection



We lose the ability to calculate ϕ_S in terms of θ . Our equations do not simplify as much. With some effort it can be shown that the ϕ_S to ϕ_I flux is $\beta\phi_I\phi_S\psi''(\theta)/\psi'(\theta)$.



Yuck?

$$\dot{\theta} = -\beta \phi_I ,$$

$$\dot{\phi}_S = -\beta \phi_I \phi_S \frac{\psi''(\theta)}{\psi'(\theta)} + \eta_1 \frac{\pi_S}{\pi} \phi_D - \eta_2 \phi_S ,$$

$$\dot{\phi}_I = \beta \phi_I \phi_S \frac{\psi''(\theta)}{\psi'(\theta)} + \eta_1 \frac{\pi_I}{\pi} \phi_D - (\eta_2 + \beta + \gamma) \phi_I ,$$

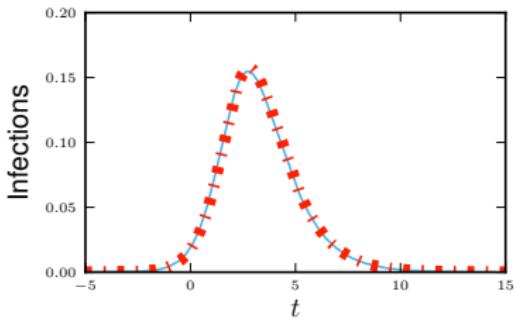
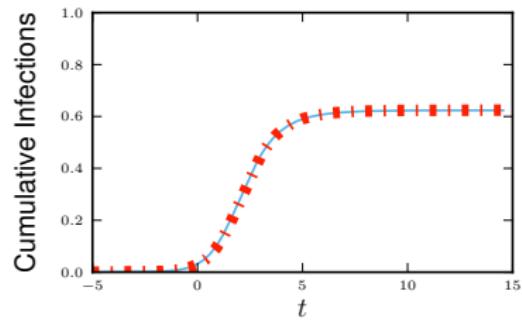
$$\dot{\phi}_D = \eta_2 (\theta - \phi_D) - \eta_1 \phi_D ,$$

$$\dot{\xi}_R = -\eta_2 \xi_R + \eta_1 \pi_R + \gamma \xi_I , \quad \xi_S = (\theta - \phi_D) \frac{\psi'(\theta)}{\psi'(1)} , \quad \xi_I = \xi - \xi_S -$$

$$\dot{\pi}_R = \eta_2 \xi_R - \eta_1 \pi_R + \gamma \pi_I , \quad \pi_S = \phi_D \frac{\psi'(\theta)}{\psi'(1)} , \quad \pi_I = \pi - \pi_S - \pi_R ,$$

$$\xi = \frac{\eta_1}{\eta_1 + \eta_2} , \quad \pi = \frac{\eta_2}{\eta_1 + \eta_2} ,$$

$$\dot{R} = \gamma I , \quad S = \psi(\theta) , \quad I = 1 - S - R .$$





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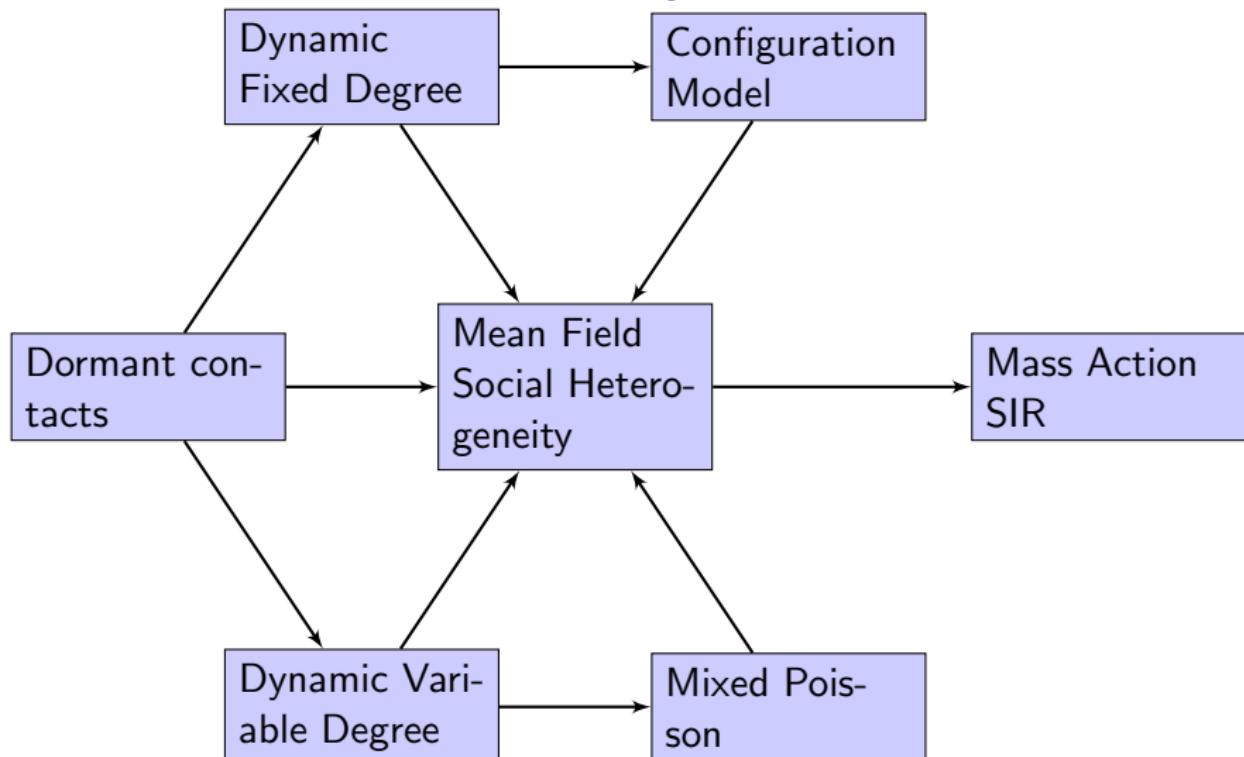
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We get

$$\mathcal{R}_0 = \frac{\beta}{\beta + \eta_2 + \gamma} \left(\frac{\langle K^2 - K \rangle}{\langle K \rangle} \frac{\eta_1}{\eta_1 + \eta_2} \frac{\eta_2 + \gamma}{\gamma} + \frac{\eta_1 \eta_2}{\gamma(\gamma + \eta_1 + \eta_2)} \right).$$

Hierarchy



General limitations for epidemics on networks

There are a number of limitations to the approaches used here.

- We do not yet understand how to handle clustering.
- Birth/death is difficult.
- Weighted edges may be tricky.
- SIS remains largely unsolved.



Networks show up in many other contexts within the study of infectious diseases.



Summary I

- The contact structure of populations regulates transmission of infectious disease.
- If contact levels are heterogeneous, the higher degree individuals play a double role in transmission — they get infected sooner and infect more people.
- For SIR diseases we can write down “simple” systems of equations that capture the dynamics of disease spread under a range of network assumptions.
- The derivation focuses on calculating the probability a random individual is susceptible, infected, or recovered.
- Clustering is difficult.
- SIS is difficult (but some decent approximations exist).



- Spread of multiple diseases is not yet well understood (but may be important).
- Networks have been measured in many relevant contexts, and the “nodes” may be individuals, cities, hospitals, etc.

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