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Random networks and epidemics

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Social networks

Two features have equal importance in disease spreading: **disease agent** (transmissability) and **social structure**

Social structure: Graph/Network: **nodes** (individuals) and **edges** ("friendship")

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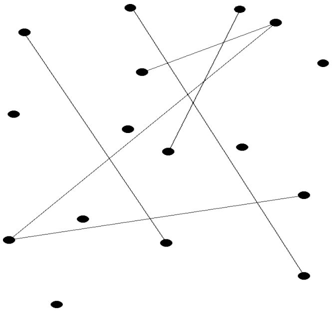

Random networks

Social structure only partly known: modelled using random graph/network **with structure**

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Some (potentially observed) local structures

- $D = \#$ friends of randomly selected individual (*degree distribution*)
- $c = P$(two friends of an individual are friends) (*clustering*)
- $\rho =$ correlation of degrees in a randomly selected friendship (*degree correlation*)
- Households (not treated further)

Other features unobserved $\Longrightarrow$ Random network

Stochastic epidemic model ön network”

Also spreading is uncertain $\Longrightarrow$ stochastic epidemic model

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Interpretation: remaining edges reflect "potential spreading"

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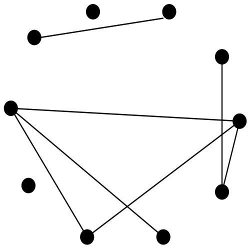
(More realistic models may have p random between different individuals and/or dependent for different friends $\Longrightarrow$ more complicated graphs)

Graph and its thinned version



Graph and its thinned version

The thinned graph is also a random graph

Those connected to index case make up final outbreak

Intervention – Control

One epidemiological reason for modelling epidemics is to understand effects of control measures.

Control measures may either aim at reducing transmission probability (vaccination, condoms, ...) and/or altering social structure (isolation, school closure, reduce travelling, ...)

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Today: "vaccination" – assuming a vaccinated person cannot get infected nor spread the disease

$\Longrightarrow$ corresponds to thinning of **nodes**

Scientific questions

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- If so, how many will get infected? (Size of giant component?)
- P(major outbreak)? (Closely related to size of giant)
- How about when vaccination is put into place?
- What is a good vaccination scheme and how many need to be vaccinated to surely avoid an outbreak?

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Probabilistic methods involved

Construction of **social network**

- Configuration model (e.g. Bollobás, 2001)
- Preferential attachment (Barabási and Albert, 1999)
- Inhomogeneous graphs (Bollobás, Janson, Riordan, 2007)
- Many different constructions!

Probabilistic methods involved

Initial phase of epidemic (also with vaccination)

Couple epidemic with "suitable" branching process:

- "giving birth" corresponds to "infecting"
- "individual" may correspond to something else (household, individual + links,...)
- R_0 = mean of offspring distribution ($R_0 > 1$ – super-critical)
- Gives P(outbreak) and relative final size (if **one** giant component)

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Extensions and additional questions

Multitype nodes: adults – children for influenza, or male – females in STIs

Multitype edges: close – distant friends, or steady – short term relationships for STIs

More general epidemic model: often leads to directed and dependent edges

Time-dynamic graphs: of interest when studying long term behaviour – endemic situations

Three subjective examples

We now present four "case studies"

The degree distribution and its effect on R_0

Britton, Janson and Martin-Löf (2007)

Model

- Social structure: Individuals have degree distribution $D \sim \{p_k\}$ and friends are chosen completely at random
- Epidemic model: each susc. friend is infected with prob p
- index case randomly selected, $n-1$ susceptible

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- $R_0 = p(E(D) - 1)$?

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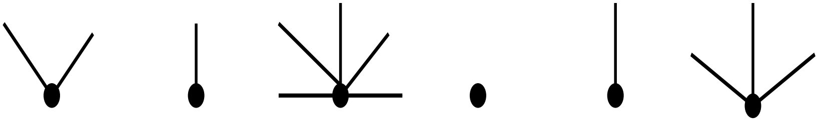
The basic reproduction number

What is the degree distribution of infectives (during early stages)?



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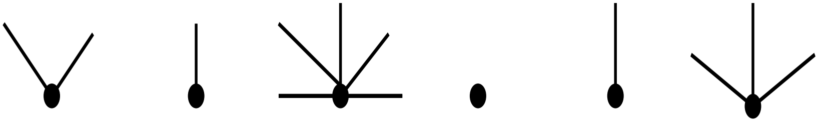

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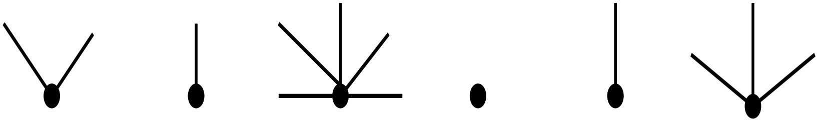

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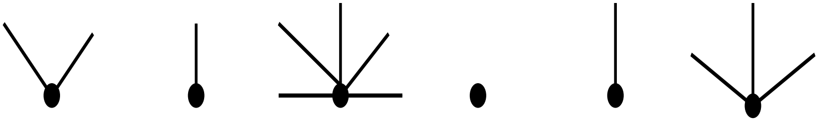

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Empirical networks have heavy-tailed degree distributions ...

The probability and size of an outbreak

The initial phase of epidemic $\approx$ branching process

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$\tau =$ fraction infected $= P(\text{random ind. belongs to giant})$

$= P(\text{index case belongs to giant}) = P(\text{major outbreak}) = \pi$

$\Longrightarrow$ outbreak size can also be derived

Vaccination

Suppose a fraction v are vaccinated prior to outbreak

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- Critical vaccination coverage: $v_{\mathrm{C}} = 1 - 1/R_0$
- **Problem**: If R_0 large (e.g. due to large $V(D)$), $v_{\mathrm{C}} \approx 1 \quad \Longrightarrow$ impossible

Vaccination, cont'd

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Vaccinees will have degree distribution $\{\tilde{p}_k\}$ rather than $\{p_k\}$

$\Longrightarrow$ much more efficient

Proportion infected as function of v, $D \sim$ Poisson

$$\left.\begin{array}{l} D \sim Po(6) \\ p = \frac{1}{2} \end{array}\right\} \Rightarrow R_0 = 3$$

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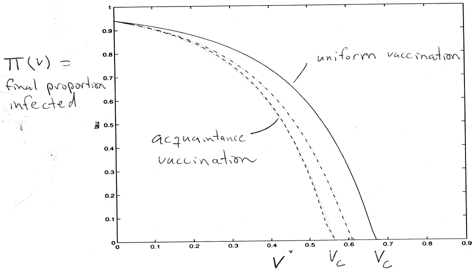

FIGURE 2. Final proportion infected τ as a function of the vaccination coverage v for four vaccination strategies: uni-

Proportion infected as function of v, $D \sim$ heavy-tailed

$D \sim$ Heavy tail ($E(D) = 6$)
$\rho = 0.5$

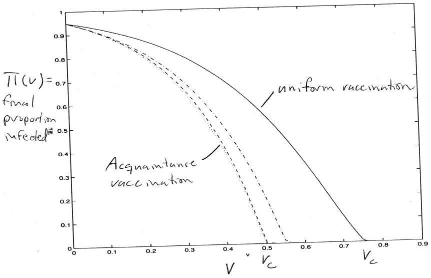

FIGURE 3. Final proportion infected as a function of the vaccination coverage for four vaccination strategies: uniform (—), acquaintance (···), E1 (- - -) and E2 (– · – · –.) The degree distribution is heavy-tailed ($p_d \propto d^{-3.5}$) with mean

Graphs with clustering

Britton, Deijfen, Lagerås and Lindholm (2008)

Random networks with clustering

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Random networks with clustering

- In many social networks (perhaps not sexual networks!) two friends of an individual are quite often friends themselves
- $c := P(\text{two friends of an individual are friends})$
- How construct a random network with predefined clustering c?

One solution: bipartite graphs

Specific construction using bipartite graphs:

1. Type 1: "true individuals" (n), Type 2: "groups" (βn)

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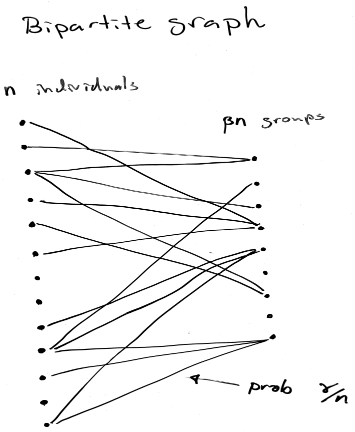
3. Project the graph on “true individuals”: individuals that share a common group are connected
4. An infected individual infects each not yet infected “friend” with prob p and then recovers.

Illustration of bipartite graph



Resulting graph:

Conclusions from analysis

- Positive clustering: $c = \frac{1}{1+\beta\gamma}$
- $E(D) = \beta\gamma^2$ (D is mixed Poisson)

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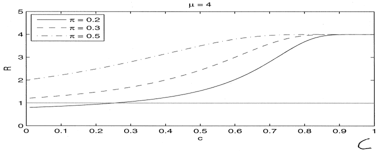
How is the epidemic affected by c?

Next slide: R_0 and P(major outbreak) as functions of c, (for fixed $E(D) = 4$ and $p = 0.2,\ 0.3,\ 0.4$)

Plot of R_0 and P(major outbreak)
as a function of c

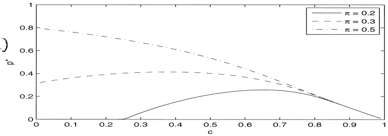
$E(D) = 4$
$p = P(\text{transmission}) = 0.2,\ 0.3,\ 0.4$

R_0



c

P(outbreak)



c

<u>Conclusions</u>

1. R_0 increases with c (surprising!)
2. P(major outbreak) may initially increase and eventually decreases.

A model for an STI in a heterosexual community

The model (Britton, Nordvik and Liljeros, 2007)

- $D = \#$ sex-partners (e.g. during a year)
- $p = P$(transmission in a relationship)

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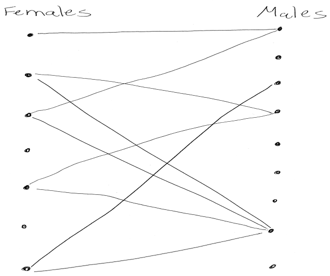

It can be shown that

$$R_0 = \sqrt{p_f \left(E(D_f) + \frac{V(D_f) - E(D_f)}{E(D_f)} \right)} \times \sqrt{p_m \left(E(D_m) + \frac{V(D_m) - E(D_m)}{E(D_m)} \right)}$$

Similar to before:

A heavy-tailed degree distribution makes R_0 large.

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\begin{aligned}
R_0 \quad &= \sqrt{p_f\left(E(D_f) + \frac{V(D_f) - E(D_f)}{E(D_f)}\right)} \\
&\times \sqrt{p_m\left(E(D_m) + \frac{V(D_m) - E(D_m)}{E(D_m)}\right)}
\end{aligned}
$$

Similar to before:

A heavy-tailed degree distribution makes R_0 large. $\Longrightarrow$

promiscuous people (super-spreaders) play an important role

Improved analysis

However:

- P(transmission) depends on # sex-acts in relationship

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- Promiscuous individuals tend to have fewer sex-acts *per partner*

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- P(transmission) depends on # sex-acts in relationship
- Promiscuous individuals tend to have fewer sex-acts *per partner*
- This should reduce R_0!

Improved analysis: continued

Extended model: short and long term relationships

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Extended model: short and long term relationships

$\Longrightarrow$ two types of edges (with different trans prob)

New (complicated) expression for R_0

The effect of different transmission probabilities depends on calibration

Calibration using survey on sexual habits

Data:

- (Anonymous) study of sexual habits in Gotland
- $\approx$ 800 people (17-28 yrs)
- Among other things: How many sex-partners during last year and how many sex-acts in each relationship

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$$P(\text{transmission}) = 1-(1-p)^{\#\ \text{sex-acts}}, \quad p = \text{per sex-act trans prob}$$

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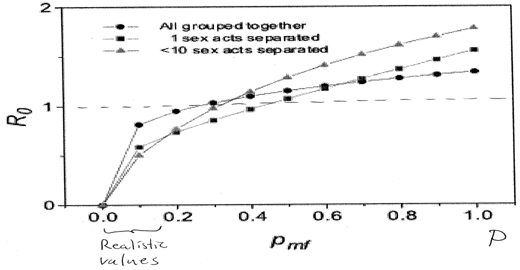
R_0 fitted to data and computed as a function of p: for one type of relationship, and two separations of short vs long

R_0 as function of p (fitted to Gotland data)

R_0 as function of $p = P(\text{transmission per contact})$

$$\left.\begin{array}{l} p_{\text{longterm}} \\ p_{\text{short term}} \end{array}\right\} = \text{mean values of} \quad 1-(1-p)^{\#\text{sex-acts}}$$

Assumption $p_m = p_f = p$



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1. Heavy-tailed degree distribution (promiscuity) increases R_0
2. Acknowledging short and long-term relationships reduces this effect
3. Endemicity not possible (for realistic p's) but maybe in sub-communities ...

Maximizing outbreak w.r.t. degree distribution

Britton and Trapman (2010)

Consider **all networks** with degree distr D with fixed mean $E(D) = \mu$ (otherwise uniform, i.e. Configuration model)

Epidemic: transmission prob p (also fixed). Random index case

Special case of interest: **Poissonian random graphs**:

- Nodes are given i.i.d. weights X_i with mean μ.
- $P(i \text{ and } j \text{ share an edge}) = X_i X_j / \mu n$
- $\Longrightarrow D \sim MixPoisson(X)$

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Question: Which distribution D or X maximizes/minimizes $\pi = P(\text{outbreak})$ and $\tau =$ size of outbreak? ($p = 1$: giant in original network)

Maximizing outbreak size w.r.t. degree distribution

Answer, Minimizing: (Easy) Choose X or D heavytailed $\Longrightarrow$ $\pi = \tau \approx 0$

Answer, Maximizing:

Poissonian random graphs:

- If $p\mu \geq \mu_c \approx 1.756$: $\pi = \tau$ are maximized by setting $X \equiv \mu$ (i.e. Erdös-Renyi-graph)

- If $p\mu < \mu_c \approx 1.756$: $\pi = \tau$ are maximized by setting $X = 0$ and $X = \mu_c/p$ (with suitable probabilities)

Maximizing outbreak size w.r.t. degree distribution

Configuration model:
- $\pi = \tau$ is maximized when D has mass only at three points: $0, k, k+1$ for some k

Intuitive explanation (for both models):
- Degree distribution should be as little random as possible
- If $p\mu$ is small enough some nodes have to be "sacrificed" (or saved) for the remaining network to be "well-connected"

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